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ORIGINAL INVESTIGATIONS

OF

CATTLE DISEASES

IN

NEBRASKA.

Revised and Augmented according to latest Investigations.

BY e

FRANK S. BILLINGS,

DIRECTOR OF THE PATHO-BIOLOGICAL LABORATORY OF THE STATE UNIVERSITY OF NEBRASKA.

ANIMAL DISEASE SERIES. NO. IV.

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SEPTEMBER 26, 1892.—The secretary of agriculture today proclaims to the cattle-growers of the west that the United States is free from contagious pleuro-pneumonia.

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ARTICLE I.—*The Corn-Fodder Disease in Cattle and Other Farm Animals, with Especial Relation to Contagious Pleuro-Pneumonia in American Beeves in England.* By FRANK S. BILLINGS, Director of the Patho-Biological Laboratory of the State University of Nebraska.

The name "corn-fodder disease," though not correctly expressing all the facts, has been selected for this pathologically new and scientifically unknown disease in cattle and other grazing animals because practical experience has shown that its eruption is either connected with the turning of cattle into stalk fields or feeding them on "snap corn" with the husks on in the later fall and early winter months, as a sort of a lazy man's method of feeding or gleaning up the remnants. As will be conclusively shown, and in conformity with the actual facts, it is not the corn-stalks, but the leaves and tender top-shoots, as well as the husks covering the ear, which indirectly cause the peculiar disease in cattle and horses of which we are about to treat. Any one who carefully observes the cattle when turned into such fields will see that they seldom meddle with the hard, dry, bulky stalks, but, as they pass along, that they pull off the leaves and the tender top portions of the stalks. The same complaint has also been attributed to the smut on corn; but a little reflection, combined with observation, would show that this idea is entirely groundless, as has been demonstrated by experimentation. Another equally absurd idea, to which we shall soon allude in detail, is that lack of water or salt is the cause of this malady. How long this disease has afflicted cattle in the United States it is impossible to determine, nor can we make any correct estimate at present as to the amount of loss it annually causes the farmers of the great corn-raising states of the west, though it is by no means limited to them. Still it can be safely said that this malady causes more loss in cattle to the western farmer than all other causes combined, not excepting abortion.* The more one becomes engaged in the scientific investigation of these questions the more does he become convinced of the great urgency existing for the collection of exact statistics as to the losses in our live stock annually, and disgusted with the farce played in nearly all our states by the totally incompetent lay cattle or live stock commissions, and the utter inadequacy of the present systems of education in the veterinary schools of the country to prepare men for these most responsible, and, if adequately filled, highly honorable positions. The last can only be accomplished in well organized government veterinary colleges. It should never be forgotten that it is the qualifications of the individual which make a position honorable, and not the position the individual. Persons totally ignorant of the advanced position of modern hygienic medicine, and the methods of exact scientific investigation, must, of necessity, be utterly incompetent to comprehend the true responsibilities or nature of the work connected with the preservation of our live stock from infectious diseases. As is well illustrated by the disease in question, such persons are totally dependent upon the musty and antiquated ideas of earlier writers for their knowledge, and absorb it unreflectingly as "gospel truth" according to the historical reputation of the author. They have neither the knowledge, ability, nor desire to think for themselves. To such empirics the "has been" must always be the true. They never think to weigh it by the living present. Facts presented to their eyes are utterly ignored, while the truth is entirely hidden by a mighty respect for the ignorance of the past, when covered with the glamour—sometimes, perhaps, deserved—of authority.

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* With regard to the prevention of abortion in cows, German investigators claim to have shown that the injection, under the skin, of a two per cent solution of carbolic acid, once every fourteen days during the fifth to the seventh month of pregnancy, will prevent this disaster in cows having acquired that tendency.

So much for an introduction!

The first allusion to this disease which I find in the literature at my command is an article by

PROF. JOHN GAMGEE ON THE "ILL EFFECTS OF SMUT IN THE FEED OF FARM ANIMALS," *

who says:

"The opportunity presented itself last fall for an inquiry as to the manner in which the smut which attacks plants may affect animals. The latter part of 1868 was, throughout America, very wet. A large amount of corn became smutty, that is to say, was attacked to a very serious extent by *Ustilago maidis*, and reports reached me from the west and south that cattle were dying in large numbers from a mysterious malady, the origin of which was unknown. From Mills county,

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* Report of the Commissioner of Agriculture on the Disease of Cattle in the United States, Washington, 1871, p. 78.

Iowa, I was informed late in November that about the 12th of the month there was a fall of snow six inches deep, and that the cattle, which usually run at large on the prairies, were taken in by all the better farmers who had their corn gathered, *and turned into the stalk fields. In about eight days the cattle began to die, all presenting the same symptoms.* My informant lost four out of nineteen head in fourteen days.

"Personal inquiries among gentlemen from different parts of the United States enabled me to trace the malady in western Virginia, Illinois, and the Carolinas. It is much to be regretted that accurate information as to the extent of the losses and localities affected cannot be secured.

"There are other circumstances under which cattle die from eating corn. The stalks, very late in the season, are apt to become very hard and indigestible, and without a free admixture of grass, which the early frosts kill, they are apt to cause indigestion and death. This is an observation that has often been made in America. The facts published with regard to the prevalence of a malady among cattle in America, caused by eating smutty corn, are very few. If, however, the real cause of so-called dry murrain had been correctly recorded there would be no difficulty in demonstrating that the condition of the corn-fields has had much to do with developing this disorder.

"The Department of Agriculture has received information of the death of cattle from eating smutty corn in Hampshire county, Massachusetts. Also from Whitley county, Indiana, where seven head of cattle out of fifty died, probably from smut in the corn-field in which the herd ranged.

"From Storey county, Iowa, it is reported that 'last November a disease appeared among herds recently turned into corn-stalk fields. The disease is evidently the dry murrain. Post-mortem examination showed the mucous membrane of the stomach to be highly inflamed. It is evident that the disease is generated in the stalk fields, and probably that it is produced by gorging the stomach when first turned into the stalks after being confined on the wild, frost-bitten prairie grass, and lack of sufficent water.' A few cattle died of dry murrain in Audubon county, in the same state, supposed to be caused by smut in the corn-stalks. A few head were lost from the same cause in Calhoun county, and many are reported to have died in Marshall county. We are, however, informed from Sac county that many cattle died in December, cause unknown, 'some supposed from eating smutty corn, but that has been disproved.' It is be regretted that more is not stated with regard to the reasons which led persons to doubt the effect of the smutty corn. Even in New York little credence was given to the action of smutty corn at first, but careful inquiry proved that after all it was the cause of the dry murrain in the fall of 1868. From Dakota county, Nebraska, we learn of dry murrain from the same cause, whereas from Shoshone county it is reported, and no doubt correctly, that the same disease has been noticed in cattle fed on prairie hay cut after frost."

Let us now subject these views of Mr. John Gamgee to critical analysis. It is self-evident that the above statements are all of an *a priori*—that is, without proof—character; that Mr. Gamgee had actually neither practical knowledge of, nor experience with, the disease of which he was writing. Yet it seems highly probable that he is the responsible authority for the prevalence of these ideas in the minds of the American veterinarians of to-day, and in our literature. In fact, Mr. Gamgee accepted and but repeated the idea prevailing more or less among cattlemen of the time, who, on observing disease sometimes occurring in their cattle after being turned into stalk fields in the fall and winter, and seeing, or perhaps supposing, smutty corn to be present, and nothing else to attribute the trouble to, jumped to the conclusion that the smut must of necessity be the cause. They did not, nor did Mr. Gamgee, stop and think that hundreds of cattle in adjoining fields, or those near by, were also turned into corn-stalks and at the same time of the year, and still did not become ill, nor did they, or he, go into these fields and examine for the existence of smut. A little common sense and critical consideration, combined with exact observation, should demonstrate to any practical man that dry corn-stalks of themselves could never have caused the trouble, nor could the smut. It is a well known fact that many cattle acquire a decided taste for corn smut, and even seek it out, and no evil results have ever occurred therefrom.

Now a word as to the term "dry murrain." It is one of those meaningless terms which have crept into veterinary literature from the days of crude empirical ignorance and been fostered by men whose education should have taught them better than to use it. The word "murrain" itself is an old English expression used to denote the existence of any malignant disease among a considerable number of cattle, and was once especially used in connection with the rinderpest and the contagious lung plague. It has no specific meaning. The word "dry" has been prefixed to it simply to designate the fact that the contents of the third stomach are in a more or less abnormally "dry" condition. We will refer to this question again later on. Let us now turn to Mr. John Gamgee's testimony upon the "smut" question.

SMUT NOT THE CAUSE OF THE DISEASE.

Mr. Gamgee says that he was "anxious to try some experiments on the action of pure smut on cattle. I employed a negro, in January, 1869, to go into the country and collect for me a large quantity of pure smut.

"It was rather late and the rain had washed most of it off the still standing stalks, but I obtained *forty-two pounds of excellent smut, free from adventitious matters.* On the 26th of February I purchased two cows in good health, aged about seven years. One cow was fed thrice daily one and one-half pounds of corn meal and three ounces of smut, with as much cut hay as she would eat. The second had the same allowance, but wet. On the 7th of March the amount of smut given in each feed was increased to six ounces. The cow fed on dry food lost flesh. On the 15th of March the dose of smut was increased to twelve ounces three times a day. The cow on the wet food increased in condition; the other one lost. *In three weeks the two cows consumed the forty-two pounds of smut!!* They had a voracious appetite the whole time, and the only indication of a peculiar diet was a very black color of the excrement."

"*Forty-two pounds of smut consumed by two cows in three weeks,*" *or twenty-one pounds to each cow, with no evil effects whatever!!*

Does any sane person for a moment think that any cow or steer turned into a stalk field would possibly consume twenty-one pounds of smut in three weeks' time? The smut would have to be most fearfully plentiful, and the field very large, which would yield twenty-one pounds of smut in the late fall or early winter months, and the cow most excessively busy. In fact, so busy would she have to be collecting that twenty-one pounds of smut in three weeks that she would have no time to collect any other food, and would probably die of starvation before she completed the job. If twenty-one pounds of smut had no ill effects upon either of the two cows when forcibly fed upon it, we may be very sure that smut never killed a bovine when turned into a stalk field; and hence, Mr. John Gamgee's testimony on this matter is absolutely worthless, from a positive point of view. As to "frost-bitten grasses" having killed cattle, were that a cause, every year more than half the cattle in our western states would have to die. Such statements, or reasoning, from persons claiming education and practical experience are absolutely sickening; yet they find credence in the minds of many veterinarians who, having no powers of observation or reflection, and being absolutely devoid of common sense, repeat these mythical statements in the ears of anxious cattlemen who are obliged to turn to them for advice.

It has been said that these misleading teachings of Mr. Gamgee have made their impression upon veterinarians and veterinary literature. In proof of this assertion the reader may turn to the Second Biennial Report of the late Board of Live Stock Agents of Nebraska, p. 23, where he may read:

> "CATTLE IN CORN-STALKS.
>
> "During the months of October, November, and December, in each year, many reports are received by telegraph and mail that cattle are dying of some unknown disease. The state veterinarian and assistants have investigated a great number of these reports and find that in every case the cause of trouble was the feeding of corn-stalks left standing in the field. More cattle have died of this in this state than of all other causes combined. The disease is so easily prevented by good management and proper feeding that the loss from this cause should be very small.
>
> "Corn-stalk fields are a very dangerous range for cattle, more especially so when the season has been favorable for ripening the stalks completely, and which has changed the starch and nutritious matters of the stem and leaf into an indigestible fibre, wholly valueless as food; and with a herd of cattle turned into such a field from off a dry-grass range in the late autumn there can be but one result: overgorging of matter, which cannot be digested, impaction, and the loss of many valuable animals.
>
> "The symptoms vary, but in nearly all outbreaks of this trouble some of the following are noticeable: In some places the animals are wild, with head erect and eyes protruding, and a disposition to go where they please, or to attack any one who may come in their way. Others stupid, dull, with low-hanging heads, more or less salivation, wabbling of the hind parts, knuckling of the fetlock joints behind, inability to get up when down, stumbling, great nervousness, twitching of the muscles, loss of sensation, loss of appetite, rapid breathing, quick pulse. Many die. All of these symptoms not noticeable in the same animal, but more or less of them are. As a preventive, cattle should not be turned into a field of dry stalks until after they have satisfied their hunger, and then only for a short time, particularly if the weather has been dry for some time previous. Allow plenty of salt and an unlimited quantity of water."

The above remarks are a striking example of the utter educational unfitness of the majority of the veterinarians of to-day to occupy the responsible position of sanitary officials. Accompanied by many similar clinical symptoms, we have in Nebraska, anthrax, which may occur at any season of the year, but particularly from August to October; a disease which is generally spoken of as "Hydrophobia in Cattle," and which, according to popular rumor, and often most positive assertions, is always connected with the presence of a "mad dog." There may be still another cattle disease which apparently occurs in July, August, and September, noticed later as "A Summer Septicæmia in Stock," in which the symptoms are much the same as those presented by animals having the corn-stalk disease, the pathological lesions being quite the same in many cases. The germ of this disease has been isolated and proven by us this summer, 1892.

SOME STATISTICS RELATIVE TO THE PREVALENCE OF THE CORN-STALK DISEASE.

As has been previously noticed, it has been known for years that a most serious fatality often follows turning cattle and horses into stalk fields to feed after the crop has been gathered. Exact statistics as to the prevalence of any such disease in the stock of this country cannot possibly be obtained, the means at command being entirely insufficient and there being no thoroughly organized veterinary police sanitary system in any part of the country. Some two years since the Hon. Sam. C. Bassett, the efficient secretary of the Nebraska Dairymen's Association, made very earnest endeavors to find out the loss among the cattle in the state from the corn-stalk disease, and did succeed in obtaining reliable information that the loss in the fall and winter of the year he made the attempt did exceed *one million dollars* in cattle alone. At the time in which the earlier investigations of this disease were made in Nebraska it was impossible to certainly ascertain that horses were as liable to it as cattle, but during my stay in Chicago numerous samples of blood from horses were sent me which had died in the same field with cattle, and under the same circumstances, and from this blood there was no difficulty in isolating and cultivating the same germ previously met with, and in several cases where samples of corn leaves were also sent to me the same germ was also obtained by feeding the leaves to rabbits. As an unusual interest among farmers n this disease immediately followed the discovery and demonstration of the germ by the Nebraska investigations, and as said similar material was also being repeatedly sent to the laboratory in Chicago, it excited the attention of the editors of some of the leading live stock and agricultural papers, among them Mr. A. S. Alexander, editor of the *Farmers' Review*, who kindly made every endeavor to interest the numerous correspondents of that able paper in the matter, which led to the receipt of the following reports, which were published in the *Farmers' Review* in the issue of that paper for February 5, 1890, and form the only information of the kind that has ever been collected. These statistics also show that the disease prevails in horses. Although in some ways repeating what has been said in other places in this essay, still the importance of the question to the live stock growers of the country warrants printing Mr. Alexander's introductory remarks here:

"The subjoined statistics, which we have summarized from the reports of our correspondents as to the prevalence of the corn-stalk disease over the stock-growing states of the west, must certainly give food for reflection to many of the readers of the *Farmers' Review*. As there has been no official investigation of this disease from which to gain statistical knowledge regarding its prevalence, it is self-evident that no judgment can be made as to whether it is on the increase or not, or as to its prevalence in certain fields year after year, or the effects upon it of rain, drouth, or drainage. That the existence of some cause in stalk fields, or that something causing fatal disease among stock, especially cattle, was in some way connected with their being turned into stalk fields, has for a great many years been more or less painfully apparent to those directly interested.

"When such a thing as a peculiar disease occurs without any actually known cause, it is but natural to man to first place the blame upon the things most palpable to his eye, and which in any way gave rise to suspicion that they might be the cause; so smut being not only a very common thing on corn in some fields, but also a rather unsightly object, and above all one of which very little was actually known, it was not at all strange that the cause of this disease should be attributed by many to this material.

"The next thing which seems to have attracted and still attracts very general attention was that on opening such animals the contents of the third stomach were found to be in an exceedingly dry condition; and as history has passed down to us the term of 'dry murrain' as the name of a disease, it was quite natural that the cause of this complaint should be said to be this condition of this stomach. But this was certainly the result of two things. Observers in the first place had neglected to use the thermometer and ascertain the thermal condition of the animals. Had they done so they would have found that the diseased animals presented a most abnormally high temperature, and that this rise was, as is the case in this trouble, extremely acute in its character.

"Again, had they been educated in the processes of disease and well acquainted with the results of a high temperature in acute infectious diseases of cattle, they would have known that this hard, dry condition of the contents of the third stomach was a very common occurrence in all such diseases; but, had they carefully examined the first stomach, they would have discovered that it contained an abundance of water for the demands of the animal. In fact, they should have remembered that cattle, like camels, are supplied with a gastric arrangement which enables them to take at one time a supply of water sufficient to last them several days, as well as food, without causing the serious results which would follow in any of the single stomached animals.

"The mysterious cause of this disease was, however, finally discovered to be a germ which infected the leaves of some corn—not all—by Dr. Billings, late of Nebraska, and published in a bulletin from the Experiment Station of that state, with all the scientific evidence necessary to place such an announcement upon a firm basis. Prof. T. J. Burrill, the botanist of the University of Illinois at Champaign, next described the disease and the germ, as it demonstrated itself in the growing corn, all of which has been published in a late bulletin from that university.

"With these few facts before them, and the statistics we now offer our readers, it must be apparent that we have to do with a subject of vital importance to the welfare of the stock-raising states.

"*Illinois.*

"For some reason which at present we are not in a position to explain or even theorize about, Illinois is practically free of the trouble now known as 'corn-stalk disease.' From over sixty counties comes the answer to our inquiries, 'We have no such disease with us.' In Iroquois county the disease was bad two years ago, and in Vermillion it was quite prevalent four years ago, but both counties report that the disease has now left them. In Hancock county the disease is decreasing—in fact, has practically disappeared, but in Tazewell it is reported as spreading, and ten head of cattle worth $150 have died, and thirty mares have aborted as a result of the disease. It has not been definitely shown that abortion among mares is actually due to the germ of this disease, but there are good reasons for presuming that the trouble is frequently from this cause. It is known, however, that smut on corn-stalks also causes abortion in mares. In Knox county 25 cattle have died; value, $400. In Mercer, 55 head; value, $1,000. In DeKalb, 70 head. In Henderson, 20 head; value, $500. In

Bond, Richland, Cumberland, Henry, Johnson, and Edwards counties a few cases of the disease have been noticed. In Warren 18 cattle, worth $432, have died.

"*Missouri.*

"In Gentry county 20 head of cattle have died of corn-stalk disease. In Worth county a few cattle have died, and also 10 head of horses valued at $650. A few steers have died in Benton county, while in Clinton 25 head worth $400 have died. The disease has been worse in Holt county, where 250 head of cattle valued at $3,750 have died. In Nodaway county the loss has amounted to $690, in Andrew county to $1,000, in McDonald county $100, and in Moniteau $100. These are the only counties in the state reporting loss from the disease.

"*Kansas.*

"Only eighteen counties in this state report corn-stalk disease as *not* prevalent. In the other counties a very serious state of affairs is shown by our reports. In Allen county 100 head of horses and some cattle have died. Cattle have also died in Butler county. In Clay county 500 head of cattle valued at $7,000, and 25 head of horses valued at $800, have succumbed to the disease. Cloud county has lost 200 head of cattle and the disease is spreading. In Ellsworth county two per cent of the cattle have been killed by the disease. Franklin county has lost 50 head of cattle worth $1,200. Geary county 300 cattle valued at $5,000 and 30 head of horses worth $1,000. Graham county reports a loss of $3,000 worth of cattle, and Harvey of $300 worth. In Kingman county 100 head of cattle valued at $1,200 have died, the disease having been worse than usual. A few cattle in Labette county have died, and in Lincoln county the number lost is placed at 127 head. Marion county reports a loss of cattle to the value of $1,000, and Ness county a similar amount. In Norton county $3,000 worth of cattle and $500 worth of horses have died from the disease, which has been uncommonly prevalent. In Osborne county the disease has spread rapidly, and the loss of cattle has aggregated $4,000. A few horses have also died. In Pawnee county eight calves have died. Phillips county has lost 1,000 head of cattle and 20 horses. The disease has been unusually common in the county. Our Pottawatomie correspondent estimates the loss of cattle in his county as amounting to no less than seven per cent. In Rice county 800 head of cattle have died. In Rooks county cattle to the value of $4,000 and horses to the value of $800 have been carried off by the disease, which has been seriously prevalent. Rush county has experienced a loss of $450 worth of cattle. Smith county reports a loss of 500 head of cattle valued at $4,000, and 25 horses valued at $900. One per cent of the cattle in Stafford county have died. Washington county has lost 200 head of cattle, and Trego and Woodson counties have lost quite a number.

"*Nebraska.*

"In this state the disease has caused a great amount of loss, and is spreading in Adams, Howard, Kearney, Merrick, Pawnee, and Seward counties. Our report opens with the announcement that in Adams county 700 head of cattle valued at $15,000 have died, and in addition to this there has been a loss of 50 head of horses valued at $5,000. A large number of cattle have died in Boone county, and in Furnas no less than 5 per cent of the cattle have succumbed, according to the report of our correspondent. In Cass county the loss of cattle by the disease has amounted to $3,500; in Dixon, $350; in Dodge, $1,050; in Franklin, $4,000; in Frontier, $100; in Howard, $1,500; in Johnson, $250; in Kearney, $1,500; in Hamilton, $3,600; in Holt, $250; in Pawnee, $2,000; in Platte, $150; in Seward, $1,100; in Stanton, $800; in Sarpy, $144; in Webster, $4,000; in York, $300; in Harlan, $1,500, and horses $250. In Merrick county, according to our correspondent, the disease has killed 1,000 head of cattle valued at $18,000.

"A veterinary surgeon of Ayr, Adams county, writes: 'The worst corn-stalk disease-infected field in this part of the state, and only four miles from here, is bottom land, the Little Blue river running through it. It has lain idle for two years, and the stalks this year are very great, in fact like small timber. The field contains about 30 acres, and 100 head of cattle and 20 head of horses were turned in, and in four weeks 63 head of cattle and five head of horses were dead. They were salted every morning and allowed to run to millet stacks and on four or five acres of meadow, tame grass. The cattle were all fat enough for beef with the exception of a few cows. They all showed the same symptoms, and all urinated profusely. One colt I attended urinated continually, and on opening I found the bladder very full. In a section of the country ten miles square, taking Ayr for the center, I found the losses as follows: Cattle, 143; value $2,525. Horses, seven; value, $445. This represents the losses of eighteen farmers. Considering this to be an inferior stock country (or, at least, only average through here), the above losses are too heavy for the farmers to stand.'

"*Iowa.*

"Fifty Iowa counties report that the disease is not known with them, but in many others it has been prevalent to a more or less serious degree. In Washington county 50 head of cattle have died. Monroe reports a loss of cattle amounting to $300, and of horses to the value of $1,200. The loss in Van Buren county has been very great, but our reporter does not furnish an estimate. Hundreds of cattle have died in Cherokee county, and upwards of $1,000 lost on that account. The disease has been more common than usual. In Buena Vista county, 34 head of cattle have died. Shelby county has had a very serious experience with the disease, 1,200 head of cattle valued at $12,000 having died. In Dallas county 15 cattle have died; in Warren, 50; in Muscatine, 6; in Marshall,. 6; in Sac, 30; in Iowa, 50; in Johnson, a few; in Jasper, 15; in Ida, 19; in Carroll, 35; in Sioux, 50; in Butler, a few; in Taylor, 50; in Benton, 20; in Pocahontas, 25; in Tama, 40; in Jackson, 5; in Madison, a few; and in Crawford, 75. The disease is more common or spreading in Marshall, Iowa, Crawford, Carroll, Sioux, and Tama counties.

"*Indiana.*

" This state has thus far nothing to complain of regarding the disease. All of our county reporters excepting two say that they can hear no complaints of the presence of the disease. The two exceptions are, Harrison county, where one animal worth $50 is said to have died of the corn-stalk disease, and Pulaski county, where 60 head of cattle, worth $1,200, and 30 horses, valued at $2,200, have died from the same trouble. In Harrison county many cattle are said to have died of this disease twelve years ago.

"*Minnesota.*

" In Stearns county 20 head of cattle valued at $200 have died of the disease. All the other counties heard from report the disease unknown.

" *Wisconsin.*

"This state does not suffer from the disease. As one of our correspondents puts it, 'We have silos for our corn-stalks, and that explains the absence of the disease.'

"*Dakota.*

" Only two counties in this state report corn-stalk disease as known there. In Lincoln and Beadle counties a few head of cattle have died from the disease.

"*Kentucky.*

"The disease has killed some cattle in Cumberland, Bracken, Nelson, and Butler counties. These are the only counties in the state which report the disease as known. Our Nelson county reporter writes: 'So far as I have been able to learn, there has been but one lot of cattle diseased in this section. They were on grass late in the fall and one or two of them died. They were then placed in a corn-stalk field and more of them died. One of the animals is still living, but has a running sore on his breast. All were afflicted in the same manner, and I think it is a lung and blood complaint.'

"*Michigan.*

"According to our reports from this state the disease is absolutely unknown within her borders."

"*Ohio.*

"Darke county is the only county in the state that reports the disease as known. In that county three head of cattle valued at $150 have died from corn-stalk disease."

While necessarily imperfect, the above statistics sufficiently demonstrate the wide extent of territory over which this fatal stock disease is spread, and also indicate that the actual losses must have been and are infinitely greater than those reported, as many sections of each state from which reports were received were not heard from.

The following letter from the state veterinarian of Indiana comes quite appropriate in this place, and is especially recommended to the consideration of those persons who do not believe that any germ exists in the corn:

"PRESENCE OF CORN-STALK DISEASE ILLUSTRATED BY MUZZLING ANIMALS.

"TERRE HAUTE, IND., 4–29–'91.

"That fellow lost three more animals since I saw you. He was using them in the field gathering corn. I told him to muzzle the other mules, which he did, and though they have been in the same field for three weeks since the others died not one of them has been sick.

"I made an autopsy on one of the mules that died, which, briefly stated, gave the following: lungs patchy, hypostatic congestion of left lung; broncho-pneumonia in right; five ounces serum in heart sac; mucosa of stomach patchy and inflamed; small intestines empty, hyperæmic; contents of large intestine quite dry and hard.

"Yours, M. E. KNOWLES, *D. V. S.*"

THE OUTBREAK AT FREMONT, NEB., IN 1889.

In the month of February, 1889, a very extensive outbreak of the corn-stalk disease occurred in the vicinity of Fremont, Neb., the following accounts of which appeared in the daily papers:

"FREMONT, NEB., February 7.

"John Delaney, a farmer living in Elkhorn township, five or six miles east of Fremont, is suffering the loss of a large number of cattle from his herd by a disease which puzzles the veterinarians. He has lost fifty head to date. Yesterday he determined to investigate the cause of the fatality. He consequently summoned the state veterinarians, who made an investigation. They dissected several head of dead animals and found the symptoms exactly the same in all of them. The third stomach was packed full of dry, hard food, and the surrounding organs and tissues were badly inflamed and feverish. The doctors were unable to exactly diagnose the case, but they gave it as their opinion that it was not a contagious disease. The animals when affected first will bellow in a low, hoarse manner, shaking their heads. Within twenty-four hours after this they invariably die."

The *Tribune* of Fremont, of the 8th inst., however, gives us the results of the investigation of the "Live Stock Commission" as follows:

"The state veterinarian was seen this morning. Speaking of the fatality among John Delaney's cattle, an account of which was given in the *Tribune*, he said that Mr. Delaney and many of his neighbors felt sure that the disease was a contagious one, but there was no reason or theory for any such supposition.

"'There is no contagious disease known to veterinarians which affects the third stomach of the cow,' said the doctor, 'and that was the seat of the trouble in every case examined. The third stomach, or manifold, was packed with dry food, which, taken in the fingers, crumbled like flour, and was so light it would blow away like chaff in the wind. The whole cause of the trouble is the condition of the food the cattle eat. The hay and corn-stalks are excessively dry, owing to the very dry fall and winter; and the cattle not having sufficient quantities of salt and water, congests are found. There has been some loss on the Hershey ranch, and a post-mortem examination of the animals disclosed the same conditions. Mr. Hershey had lost sixteen head at the time I was there a few days ago.'"

Mr. Delaney writes me that his actual loss was:

10 steers, valued at	$300
12 calves, valued at	120
39 cows, valued at	974
Total loss, 61 head, valued at	$1,394

It will be at once seen that the state veterinarian followed the authorities in his conclusions, but it will be shown that there were other lesions present in the cattle than that "the third stomach was the seat of trouble in every case examined." It will be shown that this condition of the third stomach was but the result and expression of another and most specific malady; that it was not diseased, but that its contents alone were the language of something else of an entirely different character.

This brings us again to that ridiculous term, "dry murrain." It will be seen by reading Mr. Gamgee's remarks, and those of the state veterinarian of Nebraska, that neither one considers the "corn-stalks" to be, of themselves, the chief cause of the malady, but that insufficiency of water supply plays a still more essential role in its etiology, to which these later authorities have added want of "salt" also.

More "pure theory," as the people call it, more unfounded *a priori* reasoning on conclusions than the above, could not possibly emanate from the human brain. A moment's reflection or an acquaintance with the anatomy and physiology of cattle should teach any one that such a "dry" condition of the third stomach is absolutely impossible in cattle were the food the chief cause of the disease. Any one should know that the gastric arrangement of bovines is similar to that of the camel, the first stomach being the receptacle for vast amounts of both food and water, thus supplying a surplus of such materials over the almost hourly demands of other animals in comparison, which fact has been taken advantage of by man in countries where extensive desert tracts prevail, barren of either food or water, the camels being able to go several days without either, but more especially water, without any intolerable inconvenience. It has often been recorded by travelers that when the water of the caravan had given out that a camel had been killed in order to get a supply of that vitally necessary material to the preservation of human life. In this regard camels and cattle are exactly alike, and under such conditions as prevail in our agricultural districts, it is beyond the range of possibility that cattle should go so long without water as to produce any effect whatever upon the dryness of the contents of the third stomach or manifold.

The dryness of the third stomach mentioned was not due to an insufficient supply of water, far less a lack of salt, but to an extremely high and prolonged rise of temperature in the animals in connection with an acute blood-infection; a septicæmia, which condition would have been at once revealed by the use of a thermometer upon some of the diseased (living) animals; and which any competent person would have immediately recognized in the character of the necroscopic lesions.

LACK OF WATER OR SALT CANNOT POSSIBLY CAUSE THE CORN-STALK DISEASE.

Having previously shown the impossibility of these two factors playing any role in the causation of this disease from theoretic, anatomical, and physiological grounds, I wish now to present practical evidence of the correctness of my assertions from the observations of a farmer in the form of one of the most remarkably intelligent and observant letters it has ever been my fortune to receive from a live stock breeder.

Mr. Samuel McKelvey, of Fairfield, Neb., wrote me under date of April 1, 1889:

"During the past fall and winter I have lost nine head of cattle from the stalk field trouble, whatever it is. I have lost all of these from one field of forty acres, and I have noticed closely, and have thought of different causes and theories, but the last one that died knocked all out of me. I am at a loss to know what the cause is, unless some disease is in the field. I will give you the particulars as near as I can, and if it is not asking too much of you, I should be very glad to hear your ideas about it.

"In November last I commenced feeding my cattle (about forty head) shock corn from this field of forty acres of which I have spoken. They then ran in a pasture of two hundred acres. One day I noticed them gathered in a bunch in the pasture, and on investigating found one of the number dead and swollen tight; it was a calf that had been weaned some three weeks, and in excellent condition. I did not examine it. In about two weeks, or about the last of November, I got the corn all gathered out of said forty acres, of which about three acres had been cut up in shock and the rest left, the corn being shucked off. On Sunday I turned my cattle in about one and a half hours in the forenoon. The balance of the day they were in a lot with straw (oat straw) in a rack, with plenty of water and salt in the yard, so they could get it if they wished. On Monday I turned them back in the forenoon, in said field about two hours. Tuesday turned them in about two hours in the forenoon and two hours in the afternoon. Wednesday morning I left home early with orders to turn the cattle in the stalks same as on the day before. My little boy went out after breakfast and found two dead and one sick. The hired man proceeded to doctor the sick one and while at work at it noticed another sick. The first one soon died. The second lived until the next day; stood up all the time; must have dropped dead off his feet. We saw him a few minutes before death standing up. They did not bloat until just at the last, and some did not bloat any. This last steer grunted and seemed to be in great misery. Of the four that died, three were yearling steers and one a small two-year-old. Now I thought if there was plenty of water in the two tanks in the yard that the larger cattle had kept these away until the dry food packed in the stomach and killed them. So I turned those that were left back the next day, two hours in the forenoon, and brought them into the yard and stayed with them until all drank; turned them back in the evening for the same time and saw that all drank when they came out; next day I turned them back, or looked them through before breakfast, and they were all right; sent the boy after breakfast to turn them into the stalk field. While in at breakfast one of the best two-year-old steers had been taken sick and left the rest that were about the straw rack, and went below the barn and laid down. In about two hours from the time the boy went to turn them into the field, I went to bring them in and water them, and on going to the barn to get a horse, noticed the one above spoken of lying below the barn almost dead. He would stretch out and tremble and grunt, and could only raise his head off the ground to his side. I sent for the cattle to the field at once, and stayed with the sick steer. When the cattle came in I counted them and found one short. I sent back to the field, and the report came back that another good two-year-old steer was almost dead in the stalk field. I went up and found him in about the same condition as the one just described, and they both died in about two hours. When I came back from the field, which was about one-third of a mile from the yard, the cattle that had been put in the yard were most all lying down. I went among them and noticed one that did not seem right. I went to him and scared him up, and it was all he could possibly do to get up; he was just taken. When he got up he went staggering off, stepping high. His limbs seemed to be numb and his sight affected. I drove him around and gave him a small dose of saltpetre. The more I drove him the better he seemed to be. He improved until he walked all right, but he seemed to be in great misery, and stood up and grunted until dark, when he laid down and died about 10 P. M. This, too, was a large two-year-old steer. *Now these were three of the very best of my cattle; and my water theory all gone up.*

"I took them out of this field and turned them into another. They cleaned it up, not any dying. Then I turned them into another, which they cleaned up without any trouble, when I turned them back into the forty-acre field in February, to clean it up, thinking the winter had by this time got away with the trouble. They ran there two or three days and one died. I then took them out of the field and kept them out, and made up my mind that I had just as well feed strychnine as leave them there."

Can more strong and positive practical testimony be given of the utter fallacy of the "dry murrain, short-of-water-and-salt hypothesis"?

Is stronger testimony necessary to show that some infecting element is in certain corn-fields?

Upon the same subject a Mr. W. E. Thorne, of Bladen, Webster county, Neb., writes under date of March 25, 1889:

"I have lost five head of cattle from running in corn-stalks this winter—two on the 26th of December, the others at various times since. The last one affected was about two weeks since, but it recovered."

As to the salt and water business he says:

"*I took especial care to salt and water my cattle, and know that there was no lack of either.*"

"After death I opened and examined the animals, but could not satisfy myself as to the cause of their death. *The first that died had no impaction of the third stomach whatever*, but in one or two of the later ones the contents were pretty dry and hard. When first taken they were very sick, and I thought there must be some substance in the stalks of a poisonous nature. The disease has been quite prevalent in various places about here, some fifty to seventy-five head having died within a radius of six to eight miles of this place."

THE DISEASE IN HORSES.

"BYRON, NEB., March 18, 1889.

"MY DEAR SIR: About the 10th of December, 1888, my brother and myself fenced off about 200 acres of stalks and turned into them a lot of colts, leaving them there but a short time each day; the stock did well until the first of January, when we lost seven colts within the same number of days. They appeared to become crazy and blind, most of them falling dead while running. We lost a great many colts in this way in Thayer county, Nebraska, and Republic county, Kansas.

"Yours, JOHN A. FISHER."

That there is a very direct connection between ensilage and a disease in horses has been known for a long time, though it is rare. The following quotation is interesting in this connection:

"WAS IT SILAGE OR SMUT THAT KILLED" THE HORSES?

"CHICAGO, ILL., March, 1889.

"*To the Farmers' Review:* The subject of feeding silage to horses that you have made so interesting in late numbers of the *Review* is one of great interest, and more so as relates to the death of some horses said to have died from the effects of the silage. The case from Kentucky, I think, will bear a little closer investigation. A private letter received by myself from a party in Kentucky who had lost twelve horses from eating silage, said the surgeons had pronounced it cerebro-spinal meningitis from silage eating. A noted veterinarian tells me that this disease is so rare that it would be impossible to have twelve or more cases closely associated, and gave it as his opinion that this silage was made from corn heavily charged with smut (ergot); and the conditions of the silage making were such that the formation of smut spores went on, permeating the entire mass; and by feeding the horses 'all that they could eat,' they were killed by paralysis of the spinal cord. That might easily be mistaken for meningitis. What makes me think this is so is from a statement in this letter to the effect that the silage was badly spoiled along the sides, and occasionally layers would be found badly damaged. This would show that there was an entry of air from some source that would cause the decay, and the smut spores, taking advantage of this, would propagate where the weather is no colder than it has been near Louisville, Ky., this winter. Hereabouts horsemen feed horses, brood mares, and colts large amounts of silage, 'not all they will eat,' but reasonable rations, and not a case have I heard of unfavorably. But in silo filling, stalks that are smut laden are pitched 'overboard,' not put in the silo, and smut balls are twisted off from less-producing ones. The idea is to have good silage go into the pits. Now possibly this smut business may not have any existence in fact, so far as it relates to this Kentucky silo, and this guess about it may be all wrong. By the way, this gentleman who writes me from Crescent Hill, Ky., about this matter, instances a neighbor of his, twelve miles distant, who has a silo, and has lost a similar number of horses from the effect of silage, and remarks that 'silage is killed in that section.' I am pleased to see Prof. Henry's remarks in connection with this matter, for what he says is authority and has great weight. His idea that a little acid in the silage makes it more favorable for digestion, recalls some experiments east this winter, when a certain amount of cider vinegar was mixed with the dry feed, and with the most beneficial and paying results. At the last silage congress in Cleveland, Dr. Ashmun, the health officer of Cleveland, testified that there was no danger or damage in a reasonable amount of acid in silage; and Dr. Stewart affirmed that 'without a certain per cent of lactic and acetic acid there could be no digestion.' JOHN GOULD."

I have personally seen quite a number of cases exactly corresponding to the above, where horses were fed on corn-fodder from a silo. This was some years ago in Massachusetts, but I had no conveniences at the time for properly investigating them.

PERSONAL EXPERIENCES WITH THE CORN-STALK DISEASE.

I much regret to have to say that I have never been able to see a single case of this disease as it occurs under natural conditions, my engagements in the laboratory, having had no competent assistance, being of such a nature, and the outbreaks occurring at such a distance from Lincoln, that it has been impossible for me to make either clinical or necroscopical examinations; hence my notes on this very important subject must of necessity be somewhat meagre and unsatisfactory, but to no one so much as to myself.

The first case with which I had any connection was of a most unique, and, at the time, unexplainable character. It remained a complete mystery to me until my investigations were completed on the Delaney outbreak at Fremont, in 1889. It was from this case that I first obtained the etiological organism, and it is only by comparison of my notes, of its method of development in and on different media, and especially that it came from cattle dying under the same conditions, that I am able to recognize it as the same disease which occurred at Fremont a little over a year subsequently.

A SINGULAR CASE OF THE CORN-STALK DISEASE AT AMES, NEB.

On January 6, 1888, Dr. W. A. Thomas, a veterinary surgeon in the employ of the then live stock commission, as inspector, came into the laboratory with two bottles, one containing the blood and the other some pieces of the organs of a steer which had died very suddenly at the feeding station of the Standard Cattle Company, at Ames, where he had been sent to inspect a number of horses on account of glanders. As to the steer, all he could say was "that it was considered well the night before—at least its condition did not attract any attention," and that it was found dead in the stable the next morning.

Knowing my interest in all such cases, the veterinarian spoken of hastily opened the animal and brought me the material mentioned, only reporting "the blood to have been fluid of a dark color, the liver swollen, anæmic, and of a dirty, yellowish-gray-brown color; the spleen much swollen and weighing five pounds, and the kidneys swollen; cortical substance gray-red and anæmic, while the medullary was bright red."

I at once made a microscopic examination of the blood by covering-glass specimens, and cannot express the astonishment and perplexity which came upon me on seeing an apparently pure condition of innumerable micro-organisms, having the polar-staining and belted appearance, as well as the size and form of those I had already discovered in the southern cattle plague, and which I had demonstrated, beyond the possibility of question, to be the specific cause of that disease. I still adhere to that position, all and any other investigators to the contrary. These phenomena of resemblance were still more strengthened by the fact that, like the etiological organism of the southern cattle plague, this germ colored much more sharply in a fuchsin solution than in either a blue or a violet, which fact is an essential point of differential value between these two organisms and that of the true swine plague, which colors more satisfactorily in regard to showing the belted condition in the blue or violet tinctions than in fuchsin. Further experience has taught me, especially since I have students with me, that the polar-staining of the three germs here mentioned is more due to a peculiar delicacy with which I personally treated them than a constant characteristic, for under tinction by the ordinary student, or the exposure others generally give such organisms, I find they stain diffusely, as clear ovoid rods, though the belt, or middle piece, can be easily made plain by after-treatment with diluted acids, or even alcohol itself—*lege artis to be sure.*

That the disease could not possibly be the southern cattle plague, seemed to be shown by the fact that all and every experience in the latter disease contradicted its appearance in our northern climate in the midst of the winter. Again, I soon found that pure cultures of this new germ were rapidly fatal to rabbits, while I have thus far found these rodents rather immune to the action of the etiological organism of the southern cattle plague; subcutaneous injections of over one half cubic centimeter will kill them in time, however. I also found essential points of differentiation in the development of the two organisms on solid media.

The wherefore of the death of the steer in so singular a manner, and among over a thousand others, all of which were presumably fed in the same way and with the same materials, was then a great mystery, but is so no longer.

At that time and in that case, so far as myself and all connected with it were concerned, but little was known of the relation of corn-fodder to disease; the only blame that article had received being for its dryness. The apparent singularity in this Ames case was the fact that "one steer" could alone be affected out of several hundred having free range of the stalk fields at the same time. Later experience and observation has taught, however, that such occurrences are quite characteristic of the disease, and that its common course is never herd or bunch-wise, but almost invariably one will be taken at a time, though only a day may separate the cases, and that the disease will frequently stop as suddenly as it began. All this is very easily understood when it is known to all that the diseased corn is seldom over a whole field, often only in a small locality in the field, and most frequently quite scattered in such places. It is even possible for only one or two diseased bunches to be in a very large field. Again, not all diseased corn, even though it presents the specific lesions, of necessity lodges the germ in a disease-producing condition itself for cattle. This most important of all facts connected with germ diseases, that specific germs are not always of necessity specifically pathogenic in every case, seems to have escaped the appreciation its common occurrence demands, and especially is this so where the non-occurrence of pathogenity in a single case can be so falsely twisted as to be used as argument against the Nebraska investigators by a lot of manifest incompetents.

Here, then, was a new disease discovered, and one belonging to the group of extra-organismal (etiologically) septicæmiæ, and caused by still another member of the ovoid-belted class of bacilli; but where the organism came from, or how the disease originated, was still a perfect mystery, which was not much cleared up by the appearance of a second outbreak in an entirely different part of the state, from which I also received material and the following communications:

THE CORN-STALK DISEASE AT CORTLAND, NEB., MARCH, 1888.

The material from this outbreak came to me in a still more unsatisfactory manner than that previously mentioned. It seems it was originally sent to the State Live Stock Commission, but as neither they nor the veterinarian were able to make any use of it, it was sent to this laboratory; and had it not had the name of "Dr. W. T. Brayton, Beatrice," on the wrapper, it would have been impossible to have traced the matter any further.

Microscopical examination of the fresh organs revealed the presence of, apparently, the same organism as had been found in the Ames case, and the inoculation of small animals enabled me to obtain pure cultures by which its identity with that germ was sufficiently demonstrated.

Upon writing to Dr. Brayton, I was favored with the following polite reply:

"BEATRICE, NEB., March 20, 1888.

"*F. S. Billings, Lincoln, Neb.*—DEAR SIR: Yours of March 18th received. The history of the cattle, as near as I can find out, is as follows: They were shipped from Osage, Iowa, about September 1, 1887, to Cortland, Neb. When starting from Osage they were in apparent good health, and at Cortland were unloaded and given into the charge a of Mr. —— to winter. The cattle were herded on high ground, getting their water from a small creek which runs through the same. As soon as cold weather commenced, they were taken about five or six miles from Cortland and put into a lot containing somewhere from five to six acres, on a creek bottom, and were allowed to run to flax straw and oat straw for feed, and getting their water from the creek which ran through the lot. I think the same one that runs through the pasture in which they were herded during the fall, and in which they are at present. In this lot was some timber (I do not know how much), which was their only protection from the weather.

"From this lot they were moved to their present location, and allowed to run to a millet stack and to some oat straw stacks. Upon March 13th I held post-mortems upon two dead animals, one a cow (in calf) and a yearling steer. The cow had died the night before, and was in fair condition as regards flesh; but the steer was poor. I found upon post-mortem: First, that the blood was of an unnatural color, and seemed to be thick. The heart had a blood clot in each side, and the posterior aorta contained a clot for about a foot from the heart. I found no lesions in the mouth; but when the stomach was reached the lining membrane of the rumen would peel off in large patches. The discharge from the bowels was a little thinner than natural, and streaked with blood. The lungs seemed in a healthy condition. [I have lately been informed that the investigators at Washington are trying to prove that this disease has no connection with corn-fodder, that it is not a septicæmia, and that it is simply an infectious pneumonia. All these objections have been answered in the text. The disease is a septicæmia, and when of short duration runs its course without any lung complications. The latter are invariably a secondary phenomenon and have no absolutely necessary connection with the disease.] The piece of lung sent was from the lung on the under side, which, I think, causes it to be so congested. The liver was about its natural size. The spleen about natural, with the exception that it looked blacker than usual.

"The history is as follows: These cattle would be all right at night but in the morning there would be one or two that could not get up, but showed no signs of pain. Some of these would get up with help for once or twice, and then die in the course of three or four days. Others would not get up at all, and die in from twelve to twenty-four hours. The trouble seems to affect the cattle in the best condition. Cows abort their calves and seem to do well after it. There are no external appearances of disease. These cattle have had no shelter this winter except the timber already spoken of.

"I saw both hogs and horses among the cattle, but was informed that there had been none of them sick. I saw some of the hogs eating a portion of a carcass of one of the cows."

Though the same micro-etiological organism had again been discovered as in the material from the steer of the Standard Cattle Company, at Ames, Neb., a few weeks previously, and its malignancy proven in this case also, still I was completely in the dark as to its source or origin, that is, in what manner the cattle obtained it. That the disease was due to feeding on food polluted with the germ, I felt convinced. I had no data up to this time pointing to corn-fodder as the cause. In fact, I doubted that whole business, though really knowing nothing about it, save that it seemed improbable that dry fodder alone, or even smut, could possibly cause it. It will be seen that Dr. Brayton does not mention corn-fodder or stalk fields in his letter, and at the time I did not know enough to be suspicious and ask any questions in that direction; but he does mention millet and hay-fodder, which opens up the question, can, or does, this germ invade these materials also? An answer can only be given by practical experience and observation, with the assistance of the scientific botanist, and the pathological investigator.

In order to show the value of scientific investigation, even when the ultimate result sought for has not been attained, let me say that all these experiences clearly taught that the specific cause was in some way connected with the food the cattle were getting; so I advised Dr. Brayton to change the same entirely, which was done with the most happy results.

CORN-STALK DISEASE AT FREMONT, NEB., FEBRUARY, 1889.

By referring to an earlier part of this report the reader will see that

"The state veterinarian (of Nebraska) visited this outbreak, and on February 7 said: 'There is no contagious disease known to veterinarians which affects the third stomach of a cow, and that was the seat of the disease in every case that I examined. The third stomach, or manifold, was packed with dry food, which, taken into the fingers, crumbled like flour.'"

The above shows a terrible degree of ignorance on the part of the state veterinarian. In the first place, the condition of the stomach named is, in a varying degree, common to every acute infectious disease in cattle, accompanied by an excessive rise of temperature; and again, as will be shown, there were essential, specific, and pathognomonic lesions in these animals of just such a disease, which the veriest tyro should not have allowed to escape his notice.

No sooner did I see the reports of this outbreak in the daily papers than the suspicion arose that it was probably the same disease from which I had previously examined material and procured the same micro-organism from Ames and Cortland. Unfortunately the legislative investigation farce of my goings and comings was on at the time and rendered a personal inspection of the outbreak entirely out of the question, hence, I dispatched the most trustworthy veterinarian at my command, to visit the outbreak and bring me such material (which he knew how to collect in a suitable manner) in sterilized bottles, with which he was supplied. As said above, I could not expect any detailed account of the gross-pathological lesions, still Dr. Thomas' very brief report of what he did see is sufficient to show that much more serious lesions were present than those reported by the state veterinarian; lesions which directly point to a malignant infectious disease that interfered most seriously with the circulation, and which must of necessity have been accompanied by an excessive rise in temperature. The animal examined was killed and immediately opened, the material at once being placed in the bottles previously mentioned. His report is as follows:

"SYMPTOMS AND POST-MORTEM CONDITIONS OF MR. JOHN DELANEY'S CATTLE; ALSO, HOW THEY HAVE BEEN CARED FOR SINCE DECEMBER 1, 1888.

"Lost 52 head up to February 7, 1889. Commenced dying five or six weeks previously.

"Mr. Delaney's herd was composed of 170 head, divided into three lots, viz., 100 cows and heifers, 46 last year's calves, and 24 fat steers.

"Deaths have occurred as follows: 7 fat steers, 8 or 9 calves, and 37 cows and heifers.

"Mr. Delaney's herd is in good condition, though a few are thin in flesh. The 100 head have been fed hay and ran in the stalk fields during the day. The fat steers have been fed corn and hay. The calves were fed millet until February 4. They had also some corn. The entire herd drank from one tank, supplied by a windmill, and all had a certain amount of salt.

"The hay feed is in very good condition. The cows also drank from the Elkhorn river when running in the field.

"The first symptoms noticed are switching the tail, some of them shivering, followed by bellowing, staring eyes, chasing pigs and chickens—in fact, almost anything that will come in their way—straining so violently that many of them evert the rectum and evacuate only a small amount of fæces, somewhat covered with mucus. They become lame, paralyzed, and usually stand until a short time before death. The strongest ones live longest, and the wildest die in the shortest time. *They are sick from eight hours to seven days.* It is in the latter that pneumonic lesions occur. The majority of them become wild or delirious and die within twenty-four hours after the first symptoms are noticed. Fifteen to twenty were shot to prevent them doing damage, and five or six did not get delirious.

"AUTOPSY.—*Pleuro-pneumonia sufficient to kill, the entire lungs being congested and the lower portion of the lobes solid; inflammation of the pleura, and about two gallons of serum in the thoracic cavity;* stomachs all in good condition; liver very firm and pale colored; gall-bladder well filled; urinary bladder filled; intestines inflamed.

"Mr. Delaney stated that in those he examined the liver looked 'half cooked or white, and the gall-bladder as large as a hog's bladder blown up.' Others stated that of those examined the majority of the gall-bladders were ruptured. In one case the rectum was divided, black and gangrenous; also a portion of the small intestine the same.

"Mr. Delaney's farm is on the Elkhorn and Platte river bottoms, about five or six miles northeast of Fremont, a short distance from the Elkhorn river."

It will now be remembered that the state veterinarian also said: "The hay and stalks are excessively dry, and the cattle not having sufficient quantities of water and salt, congests are found."

Just what "congests" mean pathologically is more than I can explain. The veterinarian sent by me was especially directed to look up the water question, and it is to be seen that in no way could the cattle have been without a sufficient supply of water to answer the requirements of nature.

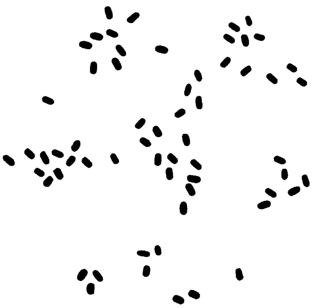
The material brought in by Dr. Thomas consisted of fresh blood from the heart, which was coagulated, the coagulum being solid and of a dark purple-red color; serum over it straw-colored and clear. There were pieces of the organs in a tin box that were somewhat frozen, for, according to my order, they were to be allowed to freeze, and kept so, immediately after being removed from the animal.

LUNG.—The pleura covering the piece of lung was much thickened, presenting an irregular, shreddy surface of a yellowish-red color, interrupted by numerous small red centers. The vessels of the interlobular spaces were engorged, much resembling the condition seen in acute stagnation pneumonia in swine plague. (See Plates I and II.) The lobuli were solidified, some being of a grayish-red color, others purplish-red, while still others were of a yellowish-gray-red color, and very anæmic. The cut surface was excessively œdematous, and the interlobular tissue was swollen. Many lobuli presented centers of a diffuse dark purple-red color, between which were others of a pearly-gray color. Others were dull gray, and still others yellowish-gray, with a varying amount of reddish tissue between them. Bronchial tubes filled with a straw-colored coagulated material. As mentioned previously, such a description would answer equally well for a form of pneumonia met with in swine plague, especially in eight or ten day cases. In fact, the structure of the lungs has such a close resemblance in cattle and swine that there is no pathologist living who could have told this piece of lung from that taken from a similar case of swine plague. This conviction would have been still more strengthened by the examination of covering-glass specimens of the tissues and the blood from this animal, from the somewhat close morphological resemblance in the germs.

LIVER.—Capsule normal; cut surface very opaque; excessively swollen and anæmic, and of a dull grayish-brown-red color; peripheral portions of the acini of yellowish-gray color; center reddish.

KIDNEYS.—Cortex much swollen, anæmic, opaque, yellowish-gray-red in color; vasa and tubuli recti much distended; medullary substance bright red, interrupted by a very pregnant distension of the tubes and an occasional large blood vessel. A small piece of the small intestine presented a very much swollen mucosa, covered with a thick viscid coating, and of a diffuse yellow-red color.

Covering-glass specimens gave apparently pure representations of one and the same organism, which corresponded exactly to those found in the Ames and Cortland outbreaks. A hanging drop, prepared directly from the blood coagulum, at once shows the organism to possess most active movements, corresponding exactly to those of the swine plague (both of which are much more active than the germs of the southern cattle plague) and possessing the same manner of development.



Germ of the corn-fodder disease.

Germ of swine plague, from a photograph by Dr. Detmers, 1888.

HISTOLOGICAL EXAMINATION OF ORGANS FROM FREMONT COW.

As is mentioned elsewhere, only a small part of the diseased organs from the cow were brought to the laboratory. This examination has, therefore, reference to sections made only from such.

LUNGS.—The lesions in this lung were of too recent origin to present any resemblance to those of contagious pleuro-pneumonia. Under low power a section from the lung presents numerous centers of consolidation, the outlines of which are more or less sharply defined from the surrounding tissue, these centers having irregular shapes and extent, the cellular products filling the alveoli being much more compact and dense in some than in others; but what is most striking is that the most intensely infiltrated alveoli are generally those most distantly situated from the bronchiole from which the irritation to the parenchymatous tissue extended. The balance of such a section presents alveoli, the majority of which are filled with a slightly granulous, very delicate, light yellowish-gray material, in which are to be seen numerous nuclei, the same being much more numerously represented in some spots than in others.

The larger vessels are compactly filled with a similar material. The outlines of each alveolus are distinctly seen and marked by nuclei-looking bodies along their course. The mucosa of the bronchioles in these sections is swollen and covered with a considerable amount of yellowish-gray material. In many alveoli the yellowish-gray material spoken of is more or less mixed up with nucleated cells. Upon closer examination with a higher power, the aforesaid yellowish-gray material in the alveoli is in many cases seen to be of an almost homogeneous character, a few delicate thread-like lines only running through it here and there. An occasional nucleated body is also to be seen. In others, on the contrary, the mass is composed of small, round cells (red blood cells), their contour being more or less distinctly marked, each cell being very clearly straw-colored, transparent, and refracting. Among these cells are to be seen numerous thread-like fibres extending in different directions, as well as quite a number of round cells, mono- and multo-nucleated, the former of which are in general much larger than the latter. The vessels taking their course along the septa of the alveoli are distended and marked by the presence of numerous round cells with distinct nuclei in close opposition with the walls, many of which are collected along the outside of the vessel in more or less extensive masses. The contents of the consolidated alveoli is made up of the usually seen round cells. The interstitial spaces appear to be completely filled up with round cell infiltration.

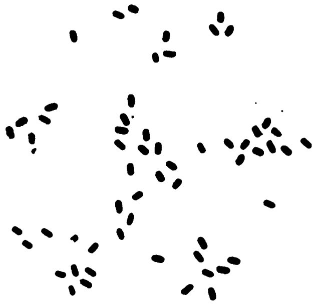
LIVER.—Offers nothing essentially striking, other than the changes upon the cells common to acute parenchymatous hepatitis. The cells are very much enlarged, many of them being but a diffuse, granulous mass, possessing nothing of a nucleated character. In others the nucleus is quite pale, the nucleolus not being at all visible, while in others again they are distinctly marked. Both around the larger and smaller blood vessels decided leucocytic migrations are seen to have taken place. In several places are circumscribed accumulations of round cells more or less toward the centre of an acinus, and lying in proximity with and in apposition with one side of a central vein, while others are more or less in the body of the acinus; but a very close examination will show that they surround or extend from a capillary.

KIDNEY.—A section of the kidney shows all the changes of acute parenchymatous nephritis. The urinary tubes are swollen, the contents of most of them being nothing but a granulous detritus. Cellular structure absent. In other tubes sometimes one swollen cell with defined outlines and prominent nucleus may be seen attached to the membrana propria, while in others several such cells may be present, but seldom in continuity. But a very few tubes, and these for but very short distances, present normal characteristics. Here and there, more or less circumscribed, but dense, round-cell infiltration may be seen to have taken place at the expense of the parenchymatous tissue, which self-evidently antedated the acute changes in the other portions. Along the line of the capillaries a limited round-cell migration has occurred, especially in points where the vessels are distended owing to compression of their lumen in the immediate vicinity, by the intensely swollen parenchyma, which led to local interference with the circulation.

EXAMINATION OF THE ORGANS FOR MICRO-ORGANISMS.

The organism described in these pages as the etiological moment in the corn-stalk disease is scattered profusely through sections of the liver, being mostly situated intercellular, that is, between the cells of the parenchyma, and plentifully represented in the blood vessels, as well as having been taken possession of in quite large numbers by an occasional round cell. In no place can they be seen to have formed threads, as is the case with its near relative, the germ of swine plague, more especially in the kidney. Occasionally two or three individuals can be seen linked together.

The above description will answer for the dispersion of the organism in the kidneys, but it may as well be mentioned that they are numerously present in the detritus of the urinary tubes; when the section has been appropriately met, the object can be seen in great numbers within the capillary loops of the Malpighian tufts. The swollen vessels between the capillaries are more or less filled with them. They are also very profusely represented in the lymph glands under the same conditions, but not so much so in the lungs, which, to my mind, is singular, for, as has been noticed in those of the Fremont cow, the alveoli were largely filled with blood or extravasata of a more or less hemorrhagic character; but it may have been that there had been no rupture of the walls of the vessels, and that the red cells passed out by diapedesis only, and that the micro-organisms could not under such circumstances. That infection in this disease is unequivocally *via* the digestive tract, has been already pointed out, and hence, their presence in the lungs from the respiratory tract is absolutely excluded. I must say that I was not surprised at finding so few germs in the lungs under such circumstances.



Morphological appearance of the germ of the corn-stalk disease as it appears in blood-smears when appropriately colored with methylin-blue or fuchsin.

ESSENTIAL CHARACTERISTICS OF THE DEVELOPMENT OF THE CORN-STALK DISEASE ORGANISM, AND THE POINTS OF DIFFERENTIATION BETWEEN IT AND THOSE OF SWINE PLAGUE AND THE SOUTHERN CATTLE PLAGUE IN AND UPON DIFFERENT CULTIVATING MEDIA.

The germ grows as well and more characteristically at ordinary room temperature than in thermostat.

POTATOES.—Upon the cut surface of sterilized and steamed potatoes the germ of the corn-stalk disease grows as grayish-white, somewhat elevated colonies, while that of swine plague develops in a sort of yellowish, dirty-olive color, sometimes resembling muddy coffee, and that of the southern cattle plague in primarily straw-yellow color. which sometimes assumes a slightly reddish shade. Very soft, moist potatoes will affect the growth and color. The chromogenic properties of germs are by no means constant, especially when developed on potatoes; the only reliable time to test these properties is when the cultures are very fresh and recently taken from the deceased animals; old cultures of these germs never produce the same marked shade as fresh ones. The corn-stalk germ, when obtained direct from corn, is much less markedly chromogenic than when taken from the blood of animals. Potatoes again are very uncertain media. The buried potatoes, that is, such as are kept in pits by farmers, are very acid, and developments often fail entirely upon these, even when coaxed in the thermostat. This fact might escape the attention of novices and cause them to declare these germs would not grow on potatoes. Potatoes raised on dry, sandy soil are much more reliable than those raised in the heavy soils of our western states. I have proven this by obtaining potatoes from Minnesota in winter and compared the results with those raised in Nebraska. When the potatoes are new this difference is not so marked. (See Plate III.)

WHITE OF EGGS.—The most practical way of sterilizing this material and of getting it in a convenient form is to steam the eggs until cooked hard, and then remove the requisite portion of the shell with sterilized forceps, after which they are placed in the moist chamber, prepared in the same manner as for potatoes or plate cultures. Upon this medium the corn-stalk germ from the heart's blood or organs of animals grows as clear yellow colonies, with slightly raised edges, and is to be easily distinguished from that of swine plague, which develops a somewhat oval projecting semi-fluid mass, the center being the much more prominent, and from that of the southern cattle plague, which presents a buff-colored colony. (See Plate IV.)

AGAR-AGAR.—(Plain, not glycerine.)—Upon this medium, when the surface is dry and the medium somewhat old and not accidentally moistened by the fluid in the bottom of the tube, the corn-stalk germ sometimes develops in a very marked and peculiar manner in contradistinction to the other two. In the color of the development there is nothing essentially different to be seen; but in comparison with the swine plague organism the edges of the cultures are more distinctly scolloped, the separating lines extending deep into the body of the cultures. But a marked point of difference is, that commencing from the line of inoculation the corn-stalk germ forms lines, or rays, which are finally limited by a diffuse semi-transparent border. Each of these lines is made up of individual colonies. Again, the cultures are much more dry, and less viscid, than those of swine plague, and adhere much more closely to the underlying agar; in fact, when old, it will break off in fragments in attempts at removal with the wire. As said, in order to get these effects the agar must be just right and have a dry surface. On agar there are no essential points of difference between the swine plague and southern cattle plague organism except rapidity of development and a slight variance in the irregularity of the edges of the cultures. (See Plate V.)

In and on Beef-Infusion Gelatine.—(Punctures.)—The growth in the gelatine itself of these organisms offers nothing of a differential characteristic nature; but their development upon the surface varies considerably, the germ of the corn-stalk disease developing and spreading over the surface most rapidly and with more leaf-like extensions. It is also more dry and lusterless than either of the others. That of the southern cattle plague is next in rapidity of development, but turns dark brown and produces a brownish cloud in the medium, while that of the swine plague spreads more slowly, but in a thicker mass and with more prominent edges. Both this and that of the southern cattle plague form a moist and more or less viscid colony, while the corn-stalk germ adheres to the gelatine and is inclined to break up on removal with the wire, especially if the cultures are a little old. (See Plate VI.)

Upon the Oblique Surface of Beef-Infusion Gelatine.—As neither of these organisms cause this medium to become fluid, it was quite interesting to test them on gelatine prepared in this way. The germ of swine plague here develops, or shows more of a yellowish-white shade than the others, and has a greater degree of opacity, the edges of the growth being very delicately irregular. The corn germ extends the most rapidly, and the growth is more pellucid-pearly white than the others, the edges being much more deeply scolloped. Toward the bottom of the growth a delicate, smooth, pearly edge embraces the scollops. The southern cattle plague germ develops the most slowly of all, and is of milky-white color, but less intense than that of the swine plague. The edges of the growth are more delicately irregular than in either of the others.

On Gelatine Plates.—The corn-stalk germ develops more or less beneath the surface, the colonies being at first roundish, but eventually almost almond shaped, some having more rounded ends than others; the color of the deeper-seated colonies is a kind of olive-gray, deeper in the center, while the peripheries are thinner, and hence lighter in color; when near the surface they are far less deep and more grayish in color. (See Plate VII.)

It is necessary for me to say right here that I have also isolated in one case from the lesions of the diseased corn itself and mixed with the specific germ another bacillus of the same length with rounded ends. Never, under any circumstances, have I seen it "belted," that is, with pole-ends, and an uncolored middle piece; this germ is narrower than the specific germ, that is, slimmer, and carries its straight sides nearer to each end, is not so ovoid, or bean-shaped; it does not fluidify gelatine and can easily be mistaken for the specific germ, *as it will kill rabbits occasionally, in large doses.* It is not the specific germ, and is not constantly present, and never has been found in cattle, but as it does occur as a mixture and will kill rabbits, some of the small-animals-fiend investigators may accidentally come across it and imagine they have made a great discovery.

INOCULATION EXPERIMENTS WITH PURE CULTURES OF THE CORN-STALK GERM.

So much humbuggery, so many misleading conclusions, and so many absurdly false statements have occurred from the experimental use of small animals in bacteriological investigations, especially in a certain quarter of this country, that I feel it my duty to younger and more embryonic investigators *to register a most emphatic caution against any one forming any positive conclusions as to the specific pathogenic property of a culture of any given germ from experimental results in small animals or any animals, other than the species in which a given germ causes under natural conditions a specific and well recognized disease both clinically and by pathological lesions.*

It has of late become quite a fashion to speak of germs which are *experimentally* infectious in small animals, *though not naturally infectious* in the species from which they were isolated as "*pathogenic*" in such small animals. That expression is a miserably unfortunate one. What more than "pathogenic," is the most specific infectious germ? We must use an adjective to define our meaning, and there is no better one than "experimentally pathogenic," in contradistinction to *specifically* or *naturally* pathogenic. It will not do to say one is infectious and the other pathogenic, for both forms of infection are equally so.

Small animals have their place, especially as a practical and easy method of purifying cultures, and in very rare instances have experimental diagnostic value, as the male guinea-pig and its testicle complication in glanders, but this is very rare indeed, though the cases may increase in number as experimentation advances, but the chances are very small as we have largely subjected the available small animals to our use. Their principal use is to obtain pure cultures from, when a culture has become polluted, or from foul organic materials, and save time and trouble as well as the sacrifice of the larger and more expensive animals, and at present human beings; but how much faster would we have advanced in the prevention of the diseases of our own species had we been able to freely make vicarious offerings of criminals, or unfortunates who would thus serve the only useful purpose possible in their otherwise useless and miserable lives.

The specific germ of a specific disease does not necessarily need a single transmission experiment to prove its specific etiological character.

Koch's rules are good and were essentially necessary in the early days of bacteriological investigation. They kept enthusiastic priority monomaniacs somewhere within the traces of reason. Clinical observation and pathological acumen were pretty nearly driven from the field for a time, however. The experiences of the ages counted as naught as against a culture and an inoculated mouse. Common sense has again assumed control, and the vagaries and errors of incompetent and ill-balanced enthusiasts have passed into oblivion, or are fast being forced into it.

Given a specific disease that for years, or centuries as is generally the case, has been recognized as such by a general and common clinical picture, its phenomena collectively, that has also been known by its specific lesions for the same length of time, or since pathologists began to study it, or even by the "specific" absence of such lesions (Tetanus Rabies), and in every case of such a disease after the method has been developed, given the presence of a specific (or one and the same) micro-organism, what pathological-bacteriologist desires any more evidence that that germ, always present, is the etiological moment in that disease, though perhaps he may not be able to cultivate it at once, for want of a proper method?

I am willing to admit that the exact fulfillment of the Koch rules completely fulfills all the evidence, that to isolate and cultivate the germ from every individual in which a natural disease occurs is very conclusive, *but the disease must first have been known, and the germ invariably discovered before that step is warranted;* that after that the inoculation of healthy animals of the same species, and production of the same disease in its entirety, and re-isolation and comparative cultivation of the same germ are still more conclusive; but when we cannot cultivate and carry this out it is not conclusive evidence against the specific nature of a given germ, if we can discover it in every case. Our failure to cultivate simply shows our methods are not natural enough. Investigators seem to have entirely overlooked the fact that this question of the specific germ of a specific disease is, after all, entirely a natural pathological question, and not by any means necessarily an experimental-bacteriological pathogenic phenomenon as to results in animals in which such germs do not cause a natural disease. Supposing, as is often the case, that cultures of a given germ do not produce any effect whatever when inoculated in any of the experimental animals in which such a disease does not naturally occur—for instance, the swine plague germ has no effect in dogs, does such negative evidence have any value whatever?

On the other hand, supposing that we have an adventitious germ, as a mixture, in a specific infectious disease, and on isolating it and by injecting cultures of it into the blood or lungs of the species it was found in it is found to produce pneumonic lesions, is that fact of a particle of value as evidence showing that such a germ is a specific and natural cause of disease in that species of animal, when it has never been found isolated and alone causing disease in animals of such species?

The bacteriological dentist, *Miller*, of Berlin, isolated a very large number of germs from the mouths of human beings which produced fatal disease in rabbits on experimental inoculation, and yet not one of those I have reference to has been isolated and found specifically connected with disease in man. Such experimental facts must be looked upon as instructive curiosities in bacteriology, for not one of those germs is known to naturally cause disease in rabbits. Suppose, for instance, Miller had expertmentally inoculated some of those germs into the blood or one of the visceral cavities, in pure cultures, in man and killed some individuals, that would have simply demonstrated an experimental pathogenic fact, but not a specific natural pathological phenomenon. Supposing we had the germ of contagious pleuro-pneumonia isolated and cultivated, and, as with the lung serum, could not produce that disease by subcutaneous inoculation, would that negative result be any evidence that it was not the specific germ of that disease? It would simply show that we had introduced it in a manner which was not its natural way of inducing contagious pleuro-pneumonia. We need more pathological acumen and more refined common sense, as well as greater honesty in some quarters, in these matters. We must ever bear in mind that our artificial conditions of nutrition and cultivation are seldom exactly identical with natural ones. Wherever we can, and always as far as possible, we must keep our experiments as close to nature as is practicable. For this reason I always aim to use animals for test experimentation of the same species as that in which the disease I am investigating occurs and from which a given germ has been isolated, and only to use small animals where their use can be considered a laboratory aid, or, in cases where there is no necessity of proving further the specific infectious nature of a given germ in that class of animals, in which it produces a natural disease. Most serious and erroneous conclusions can be avoided by adhering to this rule. Time and expense can also be saved. In the coming review of my work on swine plague this phase of experimentation will be considered with the utmost detail, but it will be necessary to allude to it again in connection with the corn-stalk disease.

THE EXPERIMENTAL INOCULATION OF PURE CULTURES OF THE CORN-STALK GERM FROM THE DELANEY OUTBREAK.

Small animals were first used in this case, merely to test the virulence of the cultures, purify them up, in order to compare them with the results obtained with apparently the same germ obtained from Ames and Cortland, Neb., the previous year, as up to this time there had not been conclusive evidence enough to warrant the use of cattle.

1. One full-grown buck-rabbit inoculated with a pure bouillon culture. In subcutis of right ear, two drops; in that of the inside of right flank, three divisions of a one cubic centimeter syringe. The ear was inoculated especially to see if there would any extreme degree of tumefaction follow.
2. One male full-grown guinea-pig. Three divisions of syringe in inside of right flank.
3. Mouse. One drop in the same locality.

Morning of lhe 12th. Rabbit ill; will not move unless made to; not eating; no local tumefaction. Mouse very ill; coiled up in corner of cage and only moved on disturbance. Guinea-pig same, but not so much depressed; nibbling a little food.

At 3 P. M., 12th. Mouse just died. No œdema at point of inoculation, but local vessels engorged. Mesenteric vessels engorged, and serosa of abdominal cavity swollen and glistening; muscles looked as if cooked; spleen, liver, and kidneys swollen, and very full of blood; lungs congested.

At 3:25 P. M. Rabbit dead. Skin and subcutaneous tissues at locus-inoculationis much engorged, but no œdema present; of a diffuse red color, with marked injection of larger vessels. No effects in the ear other than mentioned; no effusion in the cavities of the body; blood extremely thin and of a purple-red color; lymph gland much swollen and very juicy. *Liver* swollen; serosa normal, but the parenchyma reflecting through it was of a yellowish gray-red color; peripheries of acini yellowish; center red; cut surface very friable, opaque and anæmic; yellowish-gray in color. *Kidneys* swollen; capsule non-adherent; cortex anæmic, opaque; gray-red in color; medullary substance of a diffuse dark red color. *Spleen* swollen; pulp very juicy and soft; vessels of mesenterium engorged; those on curvature of stomach the same; mucosa of stomach swollen; covered with thick, viscid coating; diffuse bright-red in color; contents of small intestine fluid; mucosa much swollen, yellowish-red in color, and covered with a viscid coating; contents of large intestine pultaceous; mucosa not swollen; lungs engorged. Pure cultures of same germ as found in material from Fremont animal derived from each organ, and accurately tested by comparison with original cultures, as well as microscopically; same organisms in small intestines and isolated from the others present by plate cultures.

At 2 P. M., 13th. Guinea-pig dead. Lesions same as in rabbit; cultures conformable.

These experiments and cultivation comparisons demonstrated three facts of essential differential diagnostic value. Aside from those demonstrated by the cultivation experiments, these inoculations showed that the organisms in question could not be that of swine plague on account of its acute fatality.* It could not be that of the southern cattle plague because of the comparative immunity of rabbits to that germ in such small doses. It could not be the germ of the German "Wild-seuche" because of the total absence of enormous œdema which invariably follows such inoculations in that disease. It must then be a new disease.

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* The unbiased reader can certainly have no doubts in mind that the author is speaking of the corn-stalk disease germ and the reasons why it could not have been either the real swine plague germ, or that of Texas fever, or of the Wild-seuche, and yet we find the inference that I did not know what it was and that it might have been the so-called "swine plague" germ of the Agricultural Department, for in the special report on that germ we find these words: "In the *report of the corn-disease Billings states that a certain germ could not be that of swine plague (hog cholera) on account of its acute fatality.*" (P. 120.) Why not be honest, for was I not speaking of

EFFECTS OF INOCULATIONS WITH THE SAME MATERIAL IN A STEER AND HOG.

These inoculations were made on the 9th, two days previous to those in the small animals mentioned, six cubic centimeters of a pure bouillon culture being injected under the cutis in each case. The hog was not affected at all, and with that we will leave it.*

Steer calf five months old: 10th, temperature 10 A.M., 39.50° C.; 11th, temperature 10 A. M., 39.50° C.; 12th, temperature 10 A. M., 39.70° C.

Locus-inoculationis slightly swollen and hot; animal lying down and loath to move; respiration 60 a minute; pulse much accelerated; right lung solidified for the lower half; augmented vesicular respiration, with some thickening of the sound in superior half of this and whole of left lung; drinking much, but eating little.

At 9 A. M. on the 13th the temperature was 41° C.; respiration 60; pulse as before; made to rise with difficulty; right lung still more solidified and some pleuritis in lower portion; eyes wild; conjunctiva injected; yellowish material escaping from nostrils; severely constipated; fæces dark and hard; urine albuminous.

At 5 P. M. the temperature was 41.50°; manure becoming pultaceous; otherwise same.

At 9 A. M. on the 14th the temperature was 40.70° C.; condition about same; animal emaciating rapidly; discharge quite fluid, and of a yellowish color.

At 9 A. M. on the 15th the temperature was 40.70° C.; manure a little more solid; respiration somewhat easier; animal eating a little; lungs clearing up somewhat.

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the corn-stalk germ? Let me say here that I never came across that "swine plague" germ until 1890 in Chicago, and knew at once that it was a germ I had never seen until then. When rabbits receive two-drop intra-veinous injections of the true they will die as quickly as by subcutaneous injections of the pseudo swine plague organism.

* Later on, it will be shown that hogs can be killed when fed on the cadavers of cattle which ad died from the corn-stalk disease.

At noon on the 16th the temperature was 39.90° C.; evening, 39° C.; animal improving.

At noon on the 17th the temperature was 36.2° C.; evening, 36.2°. This looked like death. The animal was shivering, and terribly emaciated, and I felt sure of an autopsy in the morning; but on the 18th, morning temperature, 37° C.; evening, 37°; 19th, morning temperature, 37° C.; evening, 37.1°; 20th, morning temperature, 37.1° C.

The animal eventually recovered, though it became excessively emaciated. On the 16th I washed the inferior surface of its tail with Cor. Sub. 1:1000, and with sterilized knives opened to the local vein, allowing the blood to flow directly into a sterilized homœopathic vial. From the same I again obtained pure cultures of the germ, which were proven by cultivation and comparisons with various others, all of which were kept up on the different media mentioned until the investigations were terminated to my satisfaction.

A FEEDING EXPERIMENT WITH PURE CULTURES FROM THE FREMONT OUTBREAK.

Fed a full-grown female rabbit with pure bouillon cultures from Fremont cow, by pouring 25 cubic centimeters between the leaves of a quarter of a head of a small cabbage, two days in succession, beginning with February 14.

There was no change on the 15th or 16th, the animal eating with avidity. On the 16th it was somewhat quiet, but still ate its rations pretty well; 17th, back arched, hair bristling, sitting quiet in corner of cage, respirations somewhat increased, eating but little; 18th, not eating at all, remained in one place, back much arched, respiration very rapid.

At 9:30 A. M. on the 19th it was dead in the cage on my arrival at the laboratory. It was somewhat emaciated; musculature very pale. Peritoneum swollen and diffuse pink-red, with occasional punctiform red spots scattered through it; serosa of stomach and intestine swollen and clouded; vessels much engorged. Liver swollen, of a mottled grayish-yellow-and-red color; gall bladder distended, duct open into intestine; outside surface anæmic, opaque, and yellowish-gray-red in color; acini swollen; peripheries yellowish-gray in color; center reddish; spleen exceedingly swollen; pulp soft and juicy. Kidneys swollen; cortex opaque and anæmic, yellowish-gray in color; medulla diffuse red. Thoracic cavity contained about a tablespoonful of yellowish-red fluid; right lung attached to ribs in all directions and solid throughout; pericardial sac totally obliterated; portions of left lung solid, and red centers throughout solidified parts. Urine albuminous.

Cultures from heart, blood, spleen, liver, lungs, and kidneys contain the same germ as in previous cases, and developed in same manner.

FEEDING EXPERIMENTS WITH CORN-FODDER.

As to finding the germ of this disease, by feeding experiments in the leaves of every stalk of corn that comes from a field in which cattle have died, it would seem unreasonable to expect it, and yet because of failures of that kind objections have been raised against the validity of these experiments. Not only is every stalk not infected with this germ, but every diseased stalk, or better the leaves of it, does not lodge the germ in a condition capable of causing fatal illness in cattle. Too many practical tests have already been made by farmers in different parts of the west, not only to show that the germ is in the corn, but that it is in that corn only raised on damp, low land. I have had many samples of corn leaves sent me from fields in which stock have died in which I could not obtain the germ by feeding rabbits, while in others I have had most conclusive results. In this connection I have notes of having ten samples of leaves, large bunches sent me at one time while in Chicago, which on being fed to rabbits all proved negative, while at the same time I received one single leaf in an envelope which was eaten by two rabbits and killed them both, the specific germ being present in the blood of each animal. I have the past winter had some very disappointing and singular results, in feeding leaves from infested fields, the calves not being affected, while some rabbits died and the germ was isolated from their blood and others in the same cage remained well, calves and rabbits receiving leaves gathered on the same stalk at the same time. Such results are common to all experimenters, but it is considered criminal if I do not obtain positive results in every case. The simple fact is, that the rabbits which died ate infested leaves, the others did not. It is equally probable that the germ may not have had its virulent properties in all the corn leaves from many causes. Only lately a Mr. F. M. Rathburn, of Cambridge, Neb., told me that "I was just right on this matter; that one of his neighbors would not believe the germ business and insisted on turning his cattle into a low field where he had already lost some this winter, but that when he would turn them into the stalks on another field situated on high land on the other side of the street that the deaths would stop; that the man repeated this experiment with the same result until convinced. Mr. McKelvey's letter, in another place, gives similar evidence, and from Iowa and other states both veterinarians and farmers have written me the same thing repeatedly.

A very large cattle feeder and one of the best known men in Nebraska, Col. E. D. Webster, of Stratton, came to me early in December in quite an excited state of mind, stating that he was losing cattle out of a large herd he was feeding at Dunbar, Neb., in what he considered a most mysterious manner. On questioning him, the cerebral excitement and constipation, and sudden deaths in twenty-four to forty-eight hours, made me ask, "Colonel, are your cattle in stalks?" to which he answered, "*No; we have not gathered the corn, but are feeding snap corn,*" that is, corn snapped from the stalks with the husks on. I recommended "stopping that business and feeding old shelled corn and good hay if he had to buy it," which was done, and the deaths soon stopped, to begin again as suddenly, for the feeder thought the "snap corn racket a humbug," and being too lazy to go to town and haul shelled corn, began on the "snap corn" again and deaths again commenced. After that the "snap corn" was peremptorily stopped and no deaths occurred. Suffice to say, those cattle were never allowed into the stalk fields.

A Dr. Eisenhower, D. V. S., graduate of the Chicago Veterinary College, had exactly the same experience with "snap corn." He writes, "*Three steers were fed on snap corn, and I ordered it stopped and old shelled corn fed them, and only one died afterwards.*" *This is a strong argument for the silo or late corn-fodder.*

Whatever opposition may be brought up against the results of these investigations, which are as scientifically conclusive, I assume, as any evidence, both practical and experimental well can be, there are certain facts that no sane man will think of disputing:

First—That for years it has been the practical experience of farmers in the west, and even other parts of the country, that cattle turned into stalk fields after the corn has been gathered (especially in the months of November, December, January, and the early part of February, and occasionally the latter part of October) *do die*, and that they all present approximately the same symptoms.

Second—That this does not occur only when stock is turned into fields having a lower level, or having portions with a lower level, than the surrounding land. I do not mean meadows or flooded lands, but moist land in comparison to high dry land, the levels or draws between the rolls. The disease has never been observed to occur from stalks raised on high, dry, naturally well drained land.

These facts are thoroughly established.

We have then to do with a disease which in its occurrence is limited to a certain season of the year and to corn raised on a certain kind of land. With the aid of Mr. Duncanson, assistant to the professor of agriculture in the State University of Nebraska, such a field of corn was located at Tecumseh, Neb., the last summer, 1891, of which Mr. Duncanson gives the following description:

"*Description of Land.*—The land on which the diseased corn was found is situated in the suburbs of Tecumseh, Johnson county, Nebraska, on the first bottom of the Nemaha river; it is almost level, but of sufficient relief to insure good drainage; it lies at the extreme part of the valley from the river and directly at the foot of a sharp incline. The soil is a dark rich loam; it has been under cultivation for a number of years, and evidently has been fertilized a number of times.

"*Appearance of Corn in the Field.*—The first of a series of observations was made August 5, 1891. At this time the corn was in tassel and in silk. The stalks were dwarfed, slender, and, in general, presented an unhealthy appearance. The first two lower blades were in most cases dead, while the third and fourth blades were yellow and in a dying condition.

"Upon closer examination it was found that many of the large roots which extend downward were destroyed at from two to three inches below the surface of the ground. Between the sheaths of lower blades and the stalk was found semi-transparent gelatinous material, varying in consistency according to the amount of moisture; the texture of the inner surface of leaf-sheaths, in many cases, was almost destroyed, so much so that by slightly rubbing that portion of the sheath affected it was readily removed. The portion thus rubbed off was apparently the same as gelatinous matter mentioned above, excepting it was honey-combed by the tissues of the sheath. On the outer surface of diseased parts the gelatinous formation may be recognized, while the stalk is wet, by simply rubbing the hand over the surface. The lower joints or nodes are almost invariably more or less darkened; the discoloration becomes less and less distinct in each succeeding joint from the ground upward. On splitting numbers of the stalks open it was found that the same coloration of joints extended throughout the interior, while the space between nodes, with a few exceptions, was seemingly healthy. In a few instances the fibro-vascular bundles were of the same dark coloration from node to node. On the outer surface of blades and stalks was found numerous dark spots, varying in size from as small as could be recognized by the eye to large irregular patches; these spots are dark brown in color, and as they enlarge the central portion becomes light, presenting a dead appearance. Nothing of these dark spots was seen until August 29. Under the central portion of each of these spots was found, in greater or less quantities, this same gelatinous formation. Some of the ears were affected while others were apparently perfectly normal. The husks have the same dark spots as the stalks and blades. Occasionally an ear was found to have died prematurely and the inner portion next to the cob contained the same gelatinous formation as found elsewhere.

"As early as November 27th many of the stalks had decayed to such an extent that they were unable to resist the action of the wind; a very slight pull on the stalks would cause many of them to break off at the top of the ground. At the point of breakage the structure of central portion of stalks was wholly or in part destroyed, while the outer parts of stalks were decayed as badly as healthy corn should be in the month of March following."

At the end of the volume will be found some very elegantly executed plates, for which I am indebted to Professor Bessey, of the State University, illustrating the appearance of the diseased corn, which, in connection with the very clear description of Mr. Duncanson, and the later one from Burrill, of Illinois, should enable any farmer to recognize it. I was in hopes to have had a very exact description of a scientific character of the microscopic and macroscopic lesions in the corn, describing the disturbance of the various tissues, which would have marked an advance in our knowledge of this disease, but my endeavors were unsuccessful, and all I can do is to refer the reader to the plates in question.

"Plate VIII shows the small discolored spots on the leaf-sheaths.

"Plate IX shows a more advanced condition, and also the lesion at the joints at the base of the leaf-sheaths.

"Plate X represents a diseased stalk split lengthwise, showing the discolored joint and the extension of the discoloration along one of the fibro-vascular bundles."

We have now to deal with a peculiar fact, or phenomenon, which, unless I am mistaken, and all the facts thus far known are in my favor, is most strikingly illustrative of the influence of change in nutrition on the disease-producing (in animals) property of germs. It is nothing new, so far as laboratory experiences go, for we know we can increase the virulence, or disease-producing properties, of any known specific germ by changes in the conditions under which we develop it in the laboratory. We know from practical experience that the same thing occurs under natural conditions, or else we would have infectious diseases constantly with us at all times, and in all places, and under every condition of climate or soil. We know now that this corn-stalk disease is not due to the mere turning of cattle into stalk fields alone or lack of water and salt at the same time, for its outbreaks terminate about as suddenly after the middle of February as they begin after the middle of October in certain locations, yet cattle are not any the less in the stalk fields after or prior to that date. We know again that when turned into high and dry stalk fields no fatal results follow. We also know that cattle are often fed corn-fodder in August and September, yet we never heard of a case of this disease in those months, but as we know the corn itself has lesions of the disease, is it assuming too much to assert that when fed such fodder that diseased stalks must have, now and then, and perhaps frequently, been given them?

With these striking phenomena as to the natural history of this disease in cattle it has seemed to me, ever since I began to study it, that the disease-producing properties of the corn itself (the existence of the germ having been so demonstrated that no one but an imbecile could doubt it) must be due to changes in the chlorophyl (green coloring stuff) in the leaves and other parts of such a nature as to change the germ from an inoffensive organism (to animals) to a most dangerous pathogenic (disease-producing) bacterium. This hypothesis seems to me justified. The question, then, was to ascertain if the supposition was supported by undeniable facts or not. It was for the purpose of studies in this direction that Mr. Duncanson interested himself in hunting up the field of corn described by him. About the middle of August Mr. Duncanson brought several stalks of the Tecumseh corn to the laboratory. From the lesions were made bouillon cultures, and from these numerous plates; an organism exactly resembling, morphologically and in movement, the one so often spoken of as that of the corn-stalk disease was easily isolated and predominated 50 to 1 in the colonies on the plates, corresponding therein in every particular with the corn-stalk disease germ; it did not fluidify gelatine; grew the same on agar, but more whitish in color, that is, a lighter gray; and its chromogenic properties on eggs and potatoes were not so markedly developed. Otherwise it was the same. This corn fed to rabbits and other rabbits subcutaneously inoculated with pure cultures of this one germ. *Effects entirely negative.*

September 20, 1891. Mr. Duncanson again brought me some corn from the same field. The same procedures were gone through with. *From both feeding and inoculation the rabbits were ill and off feed a few days, but eventually recovered.* The corn at this time was considerably ripened, the stalks and leaves being somewhat dry and of a yellowish shade—the chlorophyl as such no more colored the leaves. (It was in the plate cultures from this lot of corn that I isolated the organism previously alluded to, about as long as the corn-stalk disease germ, but slimmer and more straight-sided.)

December 12, 1891. Mr. Duncanson again brought me some corn from the same field, and this time quite a lot. It was all dry and crispy, so that I at once fed it to rabbits and two calves. In this experiment I was disgustedly disappointed so far as the calves were concerned. They were off feed some, were constipated, and had some rise in temperature, but were unkind enough not to sicken severely or die. With the rabbits I succeeded better; they were fed the leaves from four stalks—one died in three days, the other was not ill. From the heart's blood of the one that died the specific germ was isolated, and this time it had all the chromogenic properties it has always had when derived from animals. Later comparisons have sufficiently shown that the organism thus derived from this corn is identical with that derived from cattle of which we have had numerous examples this winter, some of the essential cases of which will be noticed later on.

As has been previously said, it is unreasonable to expect positive results in every case of this kind; sufficient practical evidence has been furnished by farmers, which is supported by a very large number of experiments with the blood from cattle and horses, to place the question of the origin of this germ in corn-fodder beyond all doubt, as demonstrated in the following very conclusive test:

A FEEDING EXPERIMENT WITH CORN-FODDER FROM FREMONT, NEB.

Immediately upon finding the same micro-organisms in the blood and organs of the cow killed at Fremont by Dr. Thomas that I had found in the same kind of material from Ames and Cortland the previous year, and feeling that there might be a hidden truth behind the dry-corn-stalk-no-water theory, I wrote to Mr. Delaney, of Fremont, to send me some of his corn-stalks, and to strip off the leaves so as to save bulk. He very fortunately misunderstood me, and sent me a small package of the stripped leaves, totally insufficient for any feeding experiment in cattle. On account of the extreme demands on my time, I let the material remain in the package, and gave no thought to it until February 26, 1889. I undid the corn leaves and, as a mere matter of curiosity, I gave the leaves to a rabbit to eat, cutting off all other feed, but giving it an abundance of fresh water twice daily. So little did I think that the leaves would lead to any positive effect in the rabbit that I gave no attention to the animal, until my man told me it was dead, on the morning of March 4. In fact, it had just died as we got to the laboratory. It had been eating green food for the past two days, but had not touched that given it on the 3d. My interest in the matter was very great, and no time was lost in making examinations and cultures. In the blood, liver, spleen, kidneys, and mesenteric lymph glands the same germ as that found in the cattle was present, so far as one could judge from a microscopical examination. Cultivation experiments gave all the points of differentiation from swine plague, which experience has shown can be relied upon. The cultures were all strictly pure, as shown by cultivations on potatoes, eggs, and in gelatine, and by microscopic examination.

The lesions in the rabbit were briefly these: Swelling of the peritoneum, with straw-colored effusion in cavity; liver excessively swollen, very friable, and almost a phosphorous liver, in portions, in its degree of fatty degeneration; spleen, same as to swelling, and pulp almost semi-fluid; kidneys, cortex almost pure yellowish-gray, opaque, and anæmic, also friable; stomach two-thirds full, mucosa intensely swollen, and covered with a thick viscid coating, beneath which the tissues were almost a diffuse purple-red; contents of small intestine fluid, mucosa very thick and swollen, and covered with a glairy, viscid coating, and anæmic, as in feeding experiments with the swine plague germ; large intestine, contents pultaceous, mucosa but little swollen, though much injected; mesenterial lymph-glands resembled the strawberry glands seen in hog cholera, excessively swollen, vessels of membrane engorged; thoracic cavity, heart, and muscles opaque and very anæmic and friable; bronchial lymph-glands swollen, diffusely red and juicy; left lung hyperæmic, right normal.

Thus it will be seen that I have followed this germ, first finding it in the cattle, and then tracing it from them to the corn, demonstrated its malignancy in that material, and, beyond that, the accidental sending to me of the leaves instead of the stalks by Mr. Delaney, and the positive result in the rabbit fed with those leaves, closely conforms to the fact that cattle when turned into such stalk fields naturally take to the leaves and tender top-shoots, leaving the hard, dry stalks more or less untouched, and hence it is from the leaves that they become infected. I believe this to be the first actual demonstration of a germ of a malignant nature infesting our grasses or grains during their development, and having disease-producing properties for certain forms of animal life when fed upon them. The question still remains open, so far as this germ is concerned, Does it also penetrate the ear of the corn? That it is in the stalks I myself have demonstrated by microscopic examinations and cultures, and practical experience has shown that it is in the husks covering the ear. *It is not disease-producing when isolated from the pith of the stalk, for when inoculated from this source in either rabbits or mice it is inactive, while from the leaves it is active, as we have seen from the feeding experiment in the black rabbit,* and others of the same kind frequently made during the past three years. This fact again opens up a still more interesting question, viz.: *Does the nature of the soil (nutrition), or the chemical properties in which these extra-organismal infectious elements primarily live, or into which they get, cause their specific infectiousness in relation to animal life?*

As previously indicated we do know that moisture exerts a favorable influence in infecting the corn, and that the chlorophyl, and perhaps some other elements in this case, of the leaves of the corn has a different chemical composition from the pith of the stalk, or even its woody covering, unless it be its extremely superficial layer; hence we must face the question, as this organism is not disease-producing when derived from the stalks according to the test made with that material, does the germ cause a chemical decomposition in the elements and juices present in the leaves, and thus itself change the character of the food offered it, or does the chlorophyl itself supply a material which causes such changes in the physiological attributes of the germs as make them disease-producing in animals for a certain length of time? It is well known that in our artificial media many germs either lose this infectious property in time or can be made to do it at our pleasure. In fact, we are able to graduate that property at will.

Again, we have to discover whether this corn germ or some similar disease-producing organism also infests grasses and other fodders such as millet, as is rather indefinitely indicated in the letter from Dr. Brayton regarding the outbreak at Cortland, Neb., to which reference has been made.

Pathological investigators cannot settle this corn disease question altogether. Having traced it directly to the corn, and demonstrated the futility of "dry murrain," impacted third stomach, corn-smut, insufficient water and salt, and what-not absurd theories as to the origin of this disease in cattle, it now remains for the botanist, or perhaps more truly, botanical mycologist, the "simon-pure" bacteriologist, to more closely trace the organism in corn. One thing is very certain—this corn disease is exceedingly local in its extension, being not only limited to certain fields but even to certain portions of such fields, and often to certain stalks in a hill, or at least to individual groups of stalks.

That much of this important work has already been done may be seen by reading the following valuable letters from Prof. Burrill, of the State University of Illinois, which finds additional support in the communication of Mr. Duncanson, published in a previous portion of this report. For sufficient reason to myself it seemed best to use Mr. Duncanson's report where I did rather than in direct connection with Burrill's letters. The farmer readers will then get the same information in two different places, on the principle that "two bites of a cherry are better than one."

"CHAMPAIGN, ILL., February 4, 1889.

"The disease you speak of in cattle is a common one in this vicinity in the fall and early winter when the animals are turned into cornstalk fields, and has occurred, I think, under other conditions. The veterinarians have pronounced it due to an impaction of the third stomach, just as your men have. I have not myself seen a post-mortem. The thing is of much interest practically and scientifically and I will look out for it. T. J. BURRILL."

"CHAMPAIGN, ILL., March 11, 1889.

"I find on comparison with my old slides that the microbe from your cultures differs mainly in its smaller size. I send you a slide taken from the diseased corn-stalks (maize). This was two years ago. I did not deem the matter sufficiently worked up to publish, but did publish the account of the organism in broom-corn and sorghum—a different thing from this in maize. This corn-stalk trouble is, as I found it, very local, occurring in a given area of a field and not elsewhere. In several instances I found it only upon low spots, and in two instances on ground that had been pond holes until the year before, when they were tiled drained. In another case a whole field of ten acres on rather sandy soil, and in clover the year before, was affected. Last year a man sent me some green stalks, which he said came from a forty-acre lot, nearly all of which failed, though soil and season seemed favorable. The stalks showed plainly enough the special trouble of which I now write. The most marked effect is to be noted in the leaf-sheaths, and if you can get a leaf culture at all from the corn-stalks, I think you can best do it by strapping off some of these sheaths and taking material from the inner surface of the tissue in corroded spots. Your cultures all seem pure, and as I now have some young corn growing, I will try the effect of the same upon it. Your germ is motile in liquids, and in this agrees with my corn microbe; neither liquefies gelatine. But I did not sufficiently study the thing at the time to be able to compare much further.

"T. J. BURRILL."

THE DISEASE IN CORN.

Burrill describes the disease in corn as follows:

"It must be premised, however, that the malady as affecting this staple crop has not been fully worked out, because for some unknown reason the attempts to communicate the disease to healthy plants by the application of culture materials containing the living organism derived from diseased plants have not been successful. I am thoroughly assured, however, that these failures are from some fault in the methods tried rather than in the want of having the true disease 'germ,' for a specific organism is too uniformly present in the affected tissues to permit us to consider it accidentally connected therewith. Moreover, the progress of the disease from cell to cell corresponds exactly with the spread of the minute organism in these tissues. It is never found far from cells which plainly show the disease characteristics, though I have succeeded in making cultures from parts appearing healthy upon the very border of the affected portions. But this only goes to show that the healthy cells are invaded before they present the characteristics of the diseased cells; that is, the organism causes the disease, not the disease the organism. There was a time, perhaps, when this latter alternative might have been seriously considered, but if so, surely the time has gone by. While no one will rest content with the simple presence of a microbe even constantly in affected tissues as full and conclusive proof that such microbe is the actual active agent in the disease, still it must be admitted that this is exceedingly strong evidence of such active agency. In the present instance I have but little hesitation in saying, without successful inoculations that the organism which has been so often found in the diseased parts of the corn plant, and which has repeatedly been obtained in a state of purity by cultivation methods, is the direct cause of the mischief observed. I am the more confident of this from the fact that but few inoculation experiments under natural conditions have been tried. Of course, continued failure of these last would shake the confidence now felt, but up to the time of this writing there is no cause for such skepticism. It should be said that although some work was done upon the disease during the two seasons of 1887 and 1888, other duties were permitted to crowd out this special investigation, except as stated below.

"Further, it must be said that, so far as I now know, the evidence that the organism found by me in diseased corn is identical with that sent to me by yourself in cultures from rabbits dead from eating suspected corn-stalks rests entirely upon their microscopical appearances. I had only mounted slides of the former for comparison, the cultures having been lost. Under the microscope they do seem to be identical. Both are actively motile, as my notes of the former examinations indicate for that direct from corn, and as is readily seen in the preparations of the living 'germs' from the rabbits. Neither, it appears, produce spores, at least, endospores. They behave the same in the staining characteristics so far as tried by me. Now, for the notes you request.

"The disease in the growing corn may commence at any time during the warm season. According to my observation, it is most likely to become noticeably apparent after mid-summer, or after the corn 'shoots,' though this is by no means always the case. Very often it occurs only upon certain pretty-clearly-marked areas or patches in the fields. I think this must be very generally the case, for only two exceptions have fallen under my observation. One of these exceptions was a field of about forty acres, the other of less size; both had fertile soil and were well cared for. Neither paid the expenses of cultivation. As generally found, the affected patches are easily recognized. The corn fails to grow as in the healthy areas. Its stunted size at once arrests the attention. Diseased stalks may, indeed, be found as large as the largest, but it is probable that these became later affected. In one instance, an area of about an acre, planted with the rest, did finely until the young corn was about six to eight inches high, after which it died so completely that the farmer replanted the patch. This latter planting did well so far as is known.

"Along with the dwarf appearance designated as common, the lower leaves prematurely die, passing through the stages of becoming yellowish-green, then yellow, then withering away. Upon closer examination it will usually be seen that there are certain spots, more especially upon the basal part of the leaf, which is wrapped closely around the stalk, having a different discoloration. These are brown-watery looking at first, then darker, and finally dead. Occasionally there are livid red spots or patches in the same situation, but I am not sure that these mean the same thing. These specially affected spots vary in size from mere points to those of several inches across, often longer, in the direction of the veins of the leaf or leaf-sheath. It is in such diseased parts that the microscopic organism believed to cause the trouble can always be easily found, and from which cultures are readily made in beef broth, the juice of the corn-stalk, etc. (See Plates VIII, IX, X at end of the book.)

"The remaining characteristic of the disease is really the one most conspicuous to those who search carefully. When the corn suffers worst from the malady in question, the roots are badly affected. Beginning with the lowest and oldest, they die and decay in the ground. At length the stalk is held upright only by the later developed 'brace' roots, and even these may slowly corrode away. Under such circumstances the affected stalks are very easily pushed over or pulled up from the little hold they have in the ground. If the plant is carefully dug and the affected roots examined even with a magnifier, no evidence can be found of the work of worms or of insects of any kind. The roots simply die, though no wounds are to be found. The external layer rots most easily and quickly, and the woody inner part may then be pulled out like a string.

"Of course where the roots are thus affected the whole plant suffers, ceases growth, fails to mature its ear; any one and every one who observes at all knows that something is the matter. If one now looks closely at the brown spots on the leaf-sheaths or roots during the first stage of disease, he will often find collections of gelatinous-like exudation. Crush a minute bit of this under a microscopical cover-glass and examine with a high power of compound microscope and the living organisms to which we ascribe the disease can be seen in innumerable numbers.

"So far as observed, corn on rich lands is more likely to suffer; not unfrequently that in low places recently broken up from the sod of wild grasses seems to be most affected. Sometimes, instead of distinct areas in a field being alone injured, scattered stalks throughout the plantation are diseased; but this seems much less common.

"The corn disease as now described is a very similar disease to that affecting broom-corn and sorghum, but is nevertheless due to a distinct organism, having at least some differences in mode of development and action under different circumstances. The broom-corn disease was deemed sufficiently understood for publication in 1887. The topic was presented by me to the meeting at Cleveland, Ohio, of the Society for the Promotion of Agricultural Science, and the paper was published in the proceedings for that year. It was also published in the transactions of the American Society of Microscopists for the same year."

RECENT INVESTIGATIONS OF THE CORN-STALK DISEASE IN CATTLE.

The Humboldt Case.

The material from these cases was sent to the laboratory October 27, 1891, during my absence, the bacteriological work was done by the then assistant in the laboratory, Dr. F. W. Brewer, which I give in his words. The experimentals in cattle and other animals with the germ isolated by Dr. Brewer were done by and under my direction and supervision. On my return to Lincoln I found a piece of cattle lung in alcohol in a bottle, which the owner assures me was from one of the animals and exactly resembles the pieces sent to Dr. Brewer and to which he refers.

The alcoholic specimen can be very briefly described, macroscopically. It represents a piece of consolidated lung about three inches long and two broad. The interstitial tissue is very strongly developed and sclerotic, the walls of the bronchial tubes are very manifest and thickened and the tubes can be seen filled with a secretion. When a sterilized wire loop is pushed into such a tube and a lot of this mass brought out and placed in caustic potash 1:10000 for a little while and then smears made from it, while some other bacilli are present, the great predominance of an ovoid belted germ is at once manifest. The "belted" appearance is only present by appropriate coloring; overexposure to the tinction at once stains the germ so that it becomes a diffusely colored ovoid bacilli, with rounded ends.

MICROSCOPIC EXAMINATION OF THE SAME TISSUES.

The interlobular tissue is densely sclerotic with very few cellular elements; more or less complete acclusion of blood vessels and lymph-sinuses; many of the large vessels have excessively thick walls, the lumen being contracted and misshapen; the lumen of such vessels is filled with a grayish cellular material in which are some leucocytes and numerous fibrin threads. The bronchial tubes generally present a very deformed appearance, the lumen being small and irregular, and filled with a dense granulous mass; epithelium either desquamated or pushed into the lumen of the cavity by a dense mass of granulation tissue which has assumed the place of the submucous reticular tissue; muscularis thickened, extending from it in all directions is a dense mass of granulous neoplastic tissue which in the line of the tubes and their immediate vicinity has become organized and sclerotic, the same has encroached largely on the parenchymatous tissue, destroying, in many places, the alveolar structure; the walls of the bronchial arteries and vessels in the parenchyma are quite thickened, their lumina unduly small and irregular in shape and in many vessels entirely destroyed; the alveolar walls are thickened and their cavities filled completely with a granulous cellular material. Sections stained for bacteria give an ovoid germ in abundance with coccoid forms and a few specimens of long, large rods.

DR. BREWER'S EXAMINATION FOR BACTERIA.

As previously remarked the owner asserts that the specimen preserved in alcohol, above alluded to, is from the lung of one of the cattle from which he sent the specimens to the laboratory which Dr. Brewer examined.

On the 27th of October, 1891, there were received at the laboratory from Mr. J. K. Cornelius, of Humboldt, Neb., a letter containing, in separate envelopes, three pieces of lung, which were from three different diseased animals, and marked respectively: No. 1, bull; No. 2, Coddington cow; No. 3, Luck cow. All the pieces were in a more or less disorganized condition on account of pressure in the mails and the process of putrefaction, the odor of which was excessively strong. Bouillon cultures were at once made by placing a small piece of each lung in flasks containing 100 cubic centimeters of beef bouillon. The flasks were all placed in a thermostat and kept at a temperature of 36.7° C.

On 30th of October a rabbit was inoculated by hypodermic injection at the inside of the hind leg with 0.7 cubic centimeter of bouillon culture No. 1.

On 1st of November, at 10:30 A. M., a rabbit was inoculated in a similar manner with 0.7 cubic centimeter of boullon culture No. 2. At the same time another rabbit was inoculated with 0.7 cubic centimeter of culture No. 3. The rabbit inoculated with 0.7 cubic centimeter of mixed bouillon culture of lung No. 1 (bull) on 30th of October at 3:30 P. M. died on 31st at 5:30 P. M., twenty-six hours after inoculation. Autopsy was made at 7 o'clock the same evening. There were not any external markings. The subcuticle vessels were intensely engorged and the peritoneal covering presented a diffuse red color with many darker petechial spots; it was also much thickened. There was also present about a teaspoonful of reddish-brown liquid mingled with froth. The visceral layer of the peritoneum was profusely red. Large intestines were of a dark leaden-gray color. The small intestines were considerably inflated and of a yellowish-gray color.

SPLEEN slightly enlarged; on the posterior border were three small patches of a dark purplish color; parenchyma rather soft.

LIVER.—At three places on the border of the left anterior lobe were several white spots nearly as large as a pea; there were also several similar spots on the right anterior lobe. Gall-bladder distended with a very black fluid.

HEART.—Anæmic; vessels engorged.

LUNGS.—Normal.

KIDNEY.—Capsule slightly adherent. Cortex reddish-brown. Parenchyma much injected, producing a diffuse red appearance.

The white and gray rabbit inoculated with 0.7 cubic centimeter of mixed bouillon culture of lung No. 2 (Coddington cow) on 1st of November at 10:30 A. M. was found dead on the morning of 3d of November at 8 o'clock; probably died about forty-five hours after inoculation. *Rigor mortis* not present. There were not any external markings. Upon making abdominal section there was observed a generally diffuse pinkish-red appearance and the larger vessels exposed were much engorged. The peritoneum appeared somewhat swollen. Mesenteric vessels and the extensions of the intestines were much engorged; a general pinkish appearance being observable. Large intestines were of a light leaden-gray color, and the duodenum of a yellowish-brownish-red. Omentum was very fatty.

SPLEEN was much enlarged; hard, swollen, and of a dark purplish color, with a few reddish-brown patches. Cut surface moist, dark purplish.

Liver.—Slightly swollen; reddish-brown in color, and there were scattered over the surface of all the lobes many white spots varying in size from that of a pin's point to a small pea. Gall-bladder full of very dark bile.

Kidney.—The exterior was of a dark brown color without any special markings; cortex reddish-brown, vessels injected; medullary portion diffusely red, vessels much engorged.

Heart.—Anæmic; vessels engorged.

Lungs.—The right superior lobe showed a commencing atalectasis in one spot.

The black rabbit inoculated with 0.7 cubic centimeter of mixed bouillon culture of lung No. 3 (Luck cow) on 1st of November at 10:30 A. M. was found dead on the morning of 4th of November at 8 o'clock; probably died about sixty-five hours after the inoculation. *Rigor mortis* marked. There were not any external markings. Upon making the cuticle incision the vessels of the subcutis were found to be much engorged in the abdominal region. At the point of inoculation and in a circle around it within the radius of about an inch the tissues were excessively red. All the vessels visible were much engorged. The peritoneal layers were somewhat diffusely reddened. Mesenteric vessels and the vessels overlying the intestines were all much engorged. Large intestines were of a grayish leaden color, and the small intestines were much distended with flatus.

Stomach was full of a watery, mucus-like material of a dark color; its mucosa was of a pinkish-gray color, and in one place at about the middle of the arc of the greater curvature, the mucosa appeared to be abraded over a somewhat circular space of about one-fourth inch in diameter, so that the serosa was quite transparent. The pyloric end of the stomach was swollen, and the annulus much so.

Spleen was small, purplish slate in color, dry and friable.

Liver was brownish-red in color, slightly darkened at the margin. Cut surface moist, slightly anæmic, vessels somewhat engorged. Gall-bladder distended with dark bile.

Kidney.—Exterior of brownish-red color, with both cortex and medullary portion light diffuse red.

Heart.—Anæmic; vessels engorged.

Lungs.—Normal, except that the left lung on the inferior margin showed a few patches of very early atelactasis. Vessels somewhat engorged.

COVER-GLASS SMEARS were made direct from the heart's blood of the rabbits and an ovoid germ was discernible, sparsely distributed.

Growth on various media as follows:

On Agar—Smoky-gray in color, raised above the surface; rapid in growth.

On Potato—Moist, slimy growth of a yellowish-brown-gray color, rapidly spreading.

On Egg—A raised, irregular light yellow growth.

Microscopical Form—An ovoid germ, which, after careful comparison with the description given by Dr. Billings in his bulletins, is I am of the opinion, the germ of the "corn-stalk" disease.

On my return from Chicago, December 1, 1891, I examined the cultures of Dr. Brewer from the inoculated rabbits and found them pure and then made comparative cultures with the germ of the corn-stalk disease obtained from rabbits fed on corn-leaves from fields where cattle had died and also from cultures from cattle that died in same fields and found all three identical and one and the same germ, and also exactly corresponding in growth with notes and descriptions of other cultures, such as those given in the first edition of this report in 1889, and other cultures obtained from cattle-blood and corn-leaves that were studied by me during my stay in Chicago in 1889 and 1890. The cultures obtained by Dr. Brewer have been repeatedly tested for virulence in rabbits, as examples:

1–7–'92.—Full-grown rabbit—died in 3 days 2 hours—¼ c. cm. pure cultures from heart's blood.

4–5–'92—Full-grown rabbit. Dose, 2 drops—death in 5 days. Pure cultures from heart's blood.

1–27–'92—Full-grown rabbit, ¼ c. cm. 1–29–'92—2 days. Pure cultures.

Intra-Jugular Inoculation of a Calf with Corn-Stalk Disease Germ.

This last rabbit was used as a control in connection with a Jersey bull calf, six months old, that received into jugular vein 9 c. cms. of a bouillon culture of Dr. Brewer's material, January 27, 1892, about 3:30 P. M.

With this calf was placed a healthy calf of about the same age, which was put in same box, and received the same food as the inoculated calf. The control calf remained well and is still well at the time of writing, April 4, 1892.

As the animal building at the experimental farm is situated two miles from the laboratory in the city I seldom see the animals experimented with but for a few moments at a time once or twice a week, my duties in the laboratory demanding more time than I can even give to them, and far less give to observing the animals also; hence my reports as to clinical phenomena must be almost null as to value, the animals being left in charge of the superintendent of the farm, who has so many other duties to perform as to be able to do little more than hastily note the temperature twice a day. In the case of this calf no temperature seems to have been observed. The inoculated calf was found dead on the morning of January 29, and word at once telephoned to the laboratory.

NECROSCOPY about 9 A. M. *Rigor mortis* well developed. No external lesions except the wound in the skin, made in exposing the jugular, which had commenced to heal *per primum* and was entirely free from suppuration. The lymph-glands in inguinal region, and especially those of mesentery, were swollen, clouded, and the vessels more or less engorged, as well as those of the mesentery itself. The serosa of the small intestines clouded, of a reddish-gray or slate color, the larger vessels being very much engorged; some spots or patches of diffuse redness were scattered here and there through the serosa. Large intestine in about the same condition externally. Lungs, in general, congested; towards the posterior edges of the inferior lobe and the anterior lobes were numerous lobuli that were somewhat below the general surface, of a diffuse darker red color, with some minute centers, tending to a grayish-red color, which were a little more prominent than the surrounding atelectatic tissue; the interlobular vessels in these portions were very markedly engorged and looked like injected vessels. A few pieces from these atelectatic centers were immediately placed in alcohol and later on studied microscopically, a description of which will follow. The heart-muscle was slightly anæmic and clouded; the left ventricle being filled with an ante-mortem clot, somewhat faded out in the ventricle, but becoming more and more red as it extended into the aorta. Blood darker than normal and coagulated more slowly, but gradually grew red on exposure to the atmosphere. Liver somewhat swollen, especially at base; acini distended, many of them anæmic and distinctly yellowish-gray in color; others brown-red. Gall-bladder distended. Cortex of kindeys somewhat anæmic and clouded, medullary substance diffuse-red, interrupted by engorgement of vasa recti, which appeared like delicate red lines extending from the apex to the base of each cone; capsule non-adherent. Spleen not much, if any, swollen, but replete in blood. Rumen normal; reticulum slightly congested; omasum about normal, contents a little dry, perhaps. Mucosa of abomasum very much swollen and gathered in marked folds of a diffuse red color, with numerous patches of a dark red scattered here and there; a viscid coating covered the entire mucosa; this stomach was almost empty; mucosa of small intestines swollen, yellow-red in duodenum and bright-red in jejunum and ileum, with many petechiæ and occasional centers of diffuse redness.

Pure cultures of the corn-stalk germ were obtained from the heart's blood of this calf and compared with other cultures from natural sources of the disease in catttle.

MICROSCOPIC EXAMINATION OF ATELECTATIC CENTERS IN LUNGS.

All of the large vessels circulating through the parenchyma of the atelectatic portions of the lungs, and the bronchial arteries as well, most exorbitantly engorged with blood, which presents itself as a cellular| grayish mass, in which are some nucleated cells and numerous fibrin threads; there is also a general engorgement of the entire vascular system and a very slight exudation into the alveoli. The mucosa of the bronchial tubes is swollen, a slight desquamation being present in some tubes; the cavities of the smaller tubes are filled with an exudate of a grayish color containing numerous nucleated cells; the submucous reticular tissues of the larger tubes contain quite a profuse representation of free nucleated cells; at the bifurcation of bronchioles, especially in the vicinity of the large veins, numerous irregularly extended accumulations of deeply tinctioned nucleated cells are present; this is also the case near the veins where no bronchiole can be seen. The interstitial or interlobular tissue is swollen, contains many free cells in its meshes; the lymph-spaces are distended and full of cellular material; the blood vessels are deeply engorged.

Sections stained for micro-organisms show ovoid-belted bacteria in apparently a pure condition in the blood-vessels and lymph-sinuses, while in the bronchial tubes, and also in the alveoli, a few adventitious germs or other organisms can be seen. The latter, or, at least, some of them, are best brought out by the Gramm method, which cannot be successfully applied to the corn-stalk disease bacterium.

FEEDING EXPERIMENTS IN THE CORN-STALK DISEASE WITH CULTURES AND ORGANS FROM INOCULATED CATTLE.

In Hogs.

Although, as mentioned in my previous report on this disease, the subcutaneous inoculation of six cubic centimeters of a pure and fresh bouillon culture of the germs failed to have any effect in the one hog subjected to this test, the calf killed by inoculation January 29, 1892, offered a good opportunity to test the value or effects of the flesh or organs of such an animal by feeding the same to hogs. Whatever we do here at the Nebraska Experiment Station, our purpose is to endeavor to obtain results of plain, every day practical value to the farmer and to leave all purely unessential things either to others or to a time when the practical necessities of the farmers have been so far met as to permit of investigations into side-issues or of possibilities of a less practical character. Laboratory experiments can as often mislead as lead in right directions, unless this practical point is kept constantly in view. It is true that apparently unessential experiments often lead, directly or indirectly, eventually to most practical results. No grain of truth is ever lost forever. At present, however, the necessities of the stock interests demand the immediate rather than the mediate facts, in so far as they can be obtained. When this calf died it was remembered that the inoculation of a hog in previous years had produced no fatal results; since then it also came to mind that the same procedure in anthrax in hogs brings no more fatal results as a general thing, but feeding anthrax bacilli or material containing those germs in almost any form, on the contrary, produces most fatal results in hogs. This fact was brought very forcibly to mind while in Chicago. A farmer lost a heifer and fed it to his hogs with very fatal results. *The heifer died of anthrax, so did the hogs that ate it up!* There is no danger whatever, for the present at least, of farmers making cultures of all sorts of germs and experimenting indiscriminately with them, *but they will feed dead cattle to hogs.* The above facts as to anthrax indicated that while one inoculation has demonstrated that that procedure might be harmless to hogs so far as the germ of corn-stalk disease is concerned, that feeding immense quantities at once might have an entirely different effect. The farmer does not care what the effects of feeding pure cultures are, and such feeding, especially in herbivora, is often very misleading as I shall show later on. What the farmer desires to know is, *is there any danger to my stock in anything I may do and should know or be taught not to do.* As said, the farmer is very liable to feed dead cattle to hogs, especially as many of them still believe in the "dry murrain," "no salt," "no water," theory as causing the corn-stalk disease. The inoculated calf gave the fitting opportunity to test this practical question. Its lungs, heart, liver, and other viscera were given to two pigs in a pen by themselves, while four other pigs receiving none (controls) were placed in two other pens respectively. The pens have asphalted floors and walls, are always scalded out and then allowed to dry and then receive a heavy coat of whitewash, and then stand dry several weeks before being used for a second experiment. The hogs had been in the pens two weeks previously. Contact with the outside world is impossible and the feeders cannot get into our animals unless so ordered. The four unfed, control hogs, remained well and are still well, April 20, 1892. One of the fed hogs died on the night of February 2, 1892, the autopsy being made on the morning of the 3d; the other fed hog became very ill, refused to eat much of anything for some time, but gradually and slowly recovered and is now with the four controls.

NECROSCOPY ON HOG FED WITH MATERIAL FROM THE INOCULATED CALF.—Five months old black and white hog, black predominating; mixed Poland China. No external markings whatever; no effusion in the visceral cavities; inguinal, mesenteric, and bronchial lymph glands somewhat swollen and clouded; mesenteric vessels pregnant and engorged. Serososa of small intestines swollen, clouded, larger vessels engorged; generally of a leaden gray color, interrupted here and there by diffuse bright red patches; serosa of large intestine the same. Spleen slightly swollen and dense; cut surface clouded and tending to dryness. Liver excessively swollen at base, edges rounded; general surface of a brownish color, with numerous acini of a yellowish-gray color interrupting it; parenchyma anæmic and dry. Kidneys slightly swollen, cortex somewhat anæmic and clouded; medullary substance bright red. Mucosa of stomach excessively swollen and gathered into deep rugæ; the mucosa of the fundus of a dark intense red color, whole mucosa covered with a viscid coating, more marked in fundus; in many places patches of a grayish granular material interrupted this coating—which were dry of themselves, and loosely attached to subtissues; mucosa of pyloric portion also much swollen, covered with a viscid coating stained yellow; mucosa of jejunum of general yellowish leaden gray color, interrupted by many patches of diffuse redness; mucosa of ileum still more swollen than that of jejunum and more of a reddish gray color; mucosa of large intestines swollen excessively; transverse folds very marked; the general appearance was of a dirty yellowish-gray, with patches of redness in the haustra; the entire surface of the mucosa was covered with a more or less closely attached dirty yellowish-gray material of a fine granular character, dry and friable in nature; when forcibly detached it exposed an injected and slightly granular surface; mucosa of rectum swollen and of a dirty yellowish-gray color, with a few petechial spots and some larger irregular shaped, diffusely extending reddish spots scattered here and there. Contents of bowels and stomach fluid. Thoracic organs comparatively normal, which shows that this germ causes a septicæmia and not necessarily pneumonia. Pure cultures were obtained from the heart's blood and by comparison determined to be the same germ as that inoculated in the calf and contained in its organs.

MORAL: *Do not feed dead stock of any kind to hogs, because: First, you may kill them; second, you may cause conditions in them which might render them unsuitable to human food and hence make them unmarketable.*

It matters not to the fact and in no way militates against the warning value of the above feeding experiment in hogs, that Nocard obtained negative results in one hog with similar material, supported by an inoculation.

Nocard says: "I have experimented in one hog only, aged three months and a quarter; it was fed the remains of all the animals used in experiment (3 pigeons, 12 rabbits, and over 20 guinea-pigs); since then it was inoculated with 2 to 3 cubic centimeters of a virulent culture. *It never appeared ill.*"

Feeding Experiments with Pure Cultures in Cattle.

Fed four months old calf 500 cubic centimeters bouillon cultures on February 2, 1892. Took temperature from that date until February 14:

February 3, 10 A. M.	39.20
February 4, 9 A. M.	39.30
February 5, 7:25 A. M.	41.70
February 5, 5:40 P. M	41.50
February 6, 7:40 A. M.	41.00
February 6, 4 P. M.	41.00
February 7, 7:50 A. M.	39.50

From this time on the temperature remained constant at about 39.50 and the animal gradually regained appetite and flesh which it had lost during the height of the rise in temperature.

CHRONIC BRONCHO-INTERSTITIAL PNEUMONIA IN CATTLE AS A SEQUEL IN THE CORN-STALK DISEASE.

SO-CALLED CONTAGIOUS PLEURO-PNEUMONIA IN AMERICAN CATTLE LANDED IN ENGLAND.

Important as are these Nebraska investigations in the nature and cause of this corn-stalk disease in our cattle to the breeder and feeder of the west, I never thought that they would be of equal, if not more importance to American cattle-growers in the bearing they would have on one of the most vital question connected with our export cattle trade; the exportation of stock cattle for feeding to Europe. As will be soon shown, however, these investigations, which, as usual, have failed to find any acknowledgment from the National Department of Agriculture, but on the contrary have met but the usual slurs and innuendoes as to their reliability, were, it seems, destined to be largely the means of solving a great international difficulty between the government of this country and that of Great Britain. The results of our investigations have been the only actual and reliable foundation furnished by the study of infectious animal diseases in this country, for comparisons by which European investigators have been enabled to remove that almost fatal stigma to our export cattle trade, the continued assertion that *contagious pleuro-pneumonia* existed in our western cattle herds and was frequently being landed in shipments of such cattle on British soil. This result alone should satisfy the stock-growers of Nebraska that they have not supported this work in vain even though we have together traveled for years through a valley of abuse and outside skepticism seldom experienced in this day and generation. Regardless of the author's connection with the work, this grand release of our western cattle from a false and politically malicious slander on their condition should encourage the stockmen of Nebraska and determine them to have these investigations continued, and to provide a suitable building for the same. It should have an equally stimulating effect on the minds of stock-growers in every other western state and inspire them to "go and do likewise," and inaugurate a similar institution and like investigations in their respective states. It should teach the Agricultural Department at Washington a most becoming lesson in modesty, and that it is more honorable as public servants to do something themselves than to persistently and constantly decry and vilify the work done in Nebraska and by other investigators. These Washington bureaucrats have, like the ancient Jews, done nothing but proclaim to the world that "nothing good can come out of the west, more particularly Nebraska." Something more was done in Nebraska in three years of investigation than has been done by the National Agricultural Department from the time it first began to investigate the diseases of live stock over twenty years ago. The reputation and work of honest investigators has never been vilified, but on the contrary supported in all the publications from the state laboratory of Nebraska.

The condemnation of American cattle in England on account of the asserted existence of contagious pleuro-pneumonia began with a shipment made in the steamship "Ontario" in 1879. At that time there was not a single ground for suspicion that the lung plague existed in any state west of the Allegheny mountains, and has not been since, with the exception of the outbreak in Chicago in 1886–87, but so well was that guarded by the Illinois Live Stock Commission that not a single case ever got outside of the quarantined limits up to the time the disease was entirely stamped out. The absolute correctness of this statement is to be seen in the fact that not a single case suspicious of the lung plague has been found in cattle either in Illinois or outside of those limits, in a western state, since then. To be sure it must be admitted that the plague still exists in New Jersey and Long Island at least, but an equal energy and honesty of purpose with that displayed by the Illinois commission by eastern authorities would have stamped it out there long ago. On the other hand, the British government has not a single just ground for condemning *western cattle* on account of the few centers of lung plague in and about eastern cities. By no possible means could western cattle destined for export come in danger of infection from such cattle, and there is no more probability of the kind of cattle diseased in those few places in the east ever being shipped west. The cattle go straight through to the steamships and directly on board of them. The western breeders do not buy scrubs or emaciated milk cows. It need not be thought that I am writing as an

American advocate, for no one living in this country, or any foreigner, is more bitterly opposed to the methods of our politicians than I am. *Free commerce in food materials, be they beef or corn, is no more in the interest of the western farmer than the wage-earners of Europe,* Protection is an insult to common sense, and a parody on every genuine humane principle.

I have placed my position thus plainly before my readers in order that they may see that no especial American sentiments control my remarks, which will be caustic, regarding the position Britain has taken on this and similar questions relative to our export trade in live stock. The government of this country is by no means blameless in this matter. The ever-increasing influence of the protection policy had to be met in some way by the British government, generally a free-trade government, and reciprocity is a two-edged sword. Politicians seldom stop at anything to carry a point, and only too often are not over-punctillious as to methods. Not until the surplus of cattle in this country forced us to seek foreign markets did the European governments, but particularly the British, find a vulnerable point in which to touch our economic vitals. Britain could neither hit our protective armor on bread-stuffs nor manufactures, except alcohol and tobacco, and the latter we cared nothing about. But she could touch the western cattle interests, plethoric as they were and still are in numbers, and hit them terribly. Not an iota more does the government of Britain really care about the wage-earners of that country than the government at Washington does for the farmers and laborers here. The landed proprietors of Britain and the protected manufacturing lords of America supply the sinews of war by which politicians live, thrive, and grow fat; both out of the life-blood of the people. Again our government was to blame. Honest scientific investigation, backed by intelligence in the secretary (it was then commissioner) of agriculture, could have as well and easily demonstrated that the supposed disease was not pleuro-pneumonia in 1880 as now. Britain could have done that also. But in regard to scientific investigation, poor as our Bureau of Animal Industry is and has been, we are still incomparably ahead of Britain. But the demonstration that the disease was not *contagious pleuro-pneumonia* in 1880 would have done no more good to our export trade to England than it will now, in 1892. The British government will shift its position. That is all! Next it will be that "*dangerous contagious disease, lump-jaw,*" and though the British sanitary authorities all admit that the meat can safely be sold for consumption "provided the diseased parts are condemned," we may yet see the British government throwing our own dirt in our faces and referring us to the authoritative opinion of the Illinois Live Stock Commission. But on the lung plague itself our skirts are by no means clear, and for "reciprocal" reasons Britain will still point her finger into the wound in our eastern side, where our authorities still nourish and protect *contagious pleuro-pneumonia,* "in order to force appropriations out of congress," as I have been told by one who should speak with authority. Verily, the live stock growers have their worst enemies in the sanitary veterinary police authorities and government officials of their own country as do the British wage-earners in similar officials in theirs.

We have not yet forgotten the sensational alarm of the existence of "*contagious foot and mouth disease*" in cattle in Missouri started by a state veterinarian official, nor how the "subsidized" British agricultural press rolled it over as a special "tid-bit" to please and support the demands of the landed proprietors of that country, as another reason for reciprocal protection against the dangers of honest commerce and cheaper food for the working classes. As in this country the veterinary sanitary officials have played into the hands of the British authorities, so there the same class of men have done their utmost to support the position taken by the government.

Three essential factors have influenced the British veterinary inspectors in asserting *contagious pleuro-pneumonia* to have been present in shipments of American cattle: First, *ignorance;* second, to keep their jobs, or Britishism *vs.* Americanism; third, jealousy of one man.

Let us consider each in its turn.

Ignorance.—That on really scientific questions, especially those of a pathological character, the British veterinary profession has been, and still is, in general, most lamentably ignorant, in comparison to that of every other European country, is a matter that cannot be disputed, and is most emphatically illustrated by the verdict in the case in point. Britain has produced but four really eminent veterinarians, Percival, Dick, Williams, and Gamgee. The "last should be first," but they "stamped him out" in England better than they ever have succeeded in doing with any animal plague, simply because of his eminent abilities beyond them all. As in this country so in Britain, the conditions of recognition offered veterinarians are not such as to attract men of the highest ability into the profession. That ignorance was a very conspicuous cause in the condemnation of American cattle is shown by a description of the lesions in the lungs of some of the cattle condemned. To be sure I have to take this description from an American source (the Report of the Department of Agriculture on "Contagious Diseases in Domestic Animals," 1880–81), but the organs described were those condemned by the veterinary inspectors of England. I do not depend at all on the verdict of the American veterinarian sent over at the time by this government, nor upon that of the quite competent pathologist, whom I personally know, that accompanied him, William F. Whitney, M. D., of Boston, though I would take his verdict on such a question as against that of all the veterinarians in Britain, but on my own judgment, based on the following descriptions and the illustrations which accompany them in the report mentioned. Why those plates should have been labeled "contagious pleuro-pneumonia," when they self-evidently never had any connection with that specific disease, and are declared not to be in the text, has been a puzzling question to me ever since I first saw them. I read the text to-day for the first time, but saw the plates in 1881. These specimens of lung are given as follows, according to name of the steamship in which the cattle were shipped:

> "*Brazilian No. 1.*—This lung contained, in about its center, a large hardened object that could be both seen and felt and would measure about six inches through its largest diameter. This, upon being cut into, appeared to be an abscess, containing nothing but a pure, rather creamy pus, and, although any portion of dead tissue that might be contained within this cavity was searched for, nothing of this sort could be found. This cavity was surrounded by what seemed to be a rather thick cartilaginous wall, this again by a considerable amount of 'marbled' tissue in which the parenchyma was of an even pinkish color, with interlobular thickening well marked, white, hard, and firm. This, in its turn, passed almost imperceptibly, the parenchyma becoming gradually more and more areable, and the intertobular thickening growing narrower and narrower into the healthy lung tissue surrounding the whole."

The British veterinary inspectors condemned that lung as being afflicted with *contagious pleuro-pneumonia;* hence, the whole shipload as equally dangerous, and the condemnation extended to the whole breed of American cattle. (I will say here, to avoid repetition, that the same action was taken in regard to the other cases that will be quoted.) The question is, was that lung affected with contagious pleuro-pneumonia? Although McFadyean, Walley, and other eminent British veterinary authorities have inferred that the writer does not know much, an opinion coincided in by the American Agricultural Department, I must say that to call such lesions as those described as representative of the lung plague would be to insult the knowledge of the veriest tyro in pathology. Neither was it an "abscess," as the very learned president of Harvard Veterinary College designates it. I cannot think for a moment that Dr. Whitney ever pronounced it such. It is as clearly a described case of *broncho-ectasis* as pen could possibly delineate, with an accumulation of muco-purulent secretion in the distension of the tube and extension of the irritation to the walls of the bronchus, thickening them by chronic irritative inflammation and further extension of the irritation to adjoining tissues, particularly the interstitial directly connected with the adventitia of the air tube and partial complication of the enclosed parenchyma. In other words, *Broncho-ectasis muco-purulenta, bronchitis parenchymatosa, peri-bronchitis; pneumonia interstitialis et parenchymatosa circumscripta.* That's all there was to it! "Enough to kill an ox," I suppose some of my farmer readers will say at the "jaw-breakers." Nevertheless, not enough to condemn either one nor all the American cattle for *Pleuro-pneumonia contagiosa.*

"*Brazilian No. 2.*—This lung, with its fellow, upon its surface presented to the eye no indication of disease, but on being pressed between the hands several small nodules within its substance at once became apparent; these, on being cut down on, in the one lung disclosed unmistakable lesions of tuberculosis, and in the other, where these indurations were much fewer and smaller, the nodules showed the peculiar lesions for which it was condemned. Several small nodules were situated in the periphery of the extreme posterior portion of the large lobe of the right lung, the larger of which was about one-half inch in diameter; in its center there appeared to be a cheesy deposit, surrounded by a very thin layer of a thin, grayish-colored pus; outside this a thin membrane, outside of which a limited amount of marbled tissue, which, near the center, was well marked, but more indistinct towards its outer margin. Of these nodules there were some four or five perfectly isolated from one another, being all, to the unaided eye, of the same description."

Is there any *contagious pleuro-pneumonia* in that description? Most assuredly not! To my mind it speaks far more for a few circumscribed centers of chronic bronchitis or bronchiolitis, with some extension to the interstitial tissues immediately adjoining, a condition that must unavoidably result in all such cases in cattle lung, and perhaps a very limited acute parenchymatous pneumonia, far more than tuberculosis. Unfortunately very few veterinarians, either in this country or Britain, are capable of differentiating between this form of isolated circumscribed bronchitis and a similar form of tuberculosis. The application of water gently poured on the cut surface of the lung will wash out the caseous secretion and expose the lumen of the tube as a small excavation, which cannot be done in case of tubercles.

"*Aleppo Lung.*—The lung from which this specimen was taken was from a bullock killed in Liverpool, July 23 and which the inspector said he considered *a fine specimen of contagious pleuro-pneumonia*, as will be seen by reference to Plate VII (Agricultural Report) which is a most perfect representation of its appearance and has very much the look of that disease, indeed so close is its resemblance that it was not until a most thorough examination had been made of the specimen that one could be warranted in saying that it was not."

That any competent pathologist should have for a moment supposed that this lesion represented a case of *contagious pleuro-pneumonia* could not be supposed for a moment. It is plainly a case of chronic circumscribed broncho-pneumonia, with some complication of the interstitial tissue in the vicinity, such as can be seen in cattle lungs very often in slaughter-houses; the acute pneumonia and congestion in the vicinity were probably caused by the irritating foul air in the "'tween decks" of the steamer. These cases will do to illustrate our point. They occurred in 1880. Such cases should not be uncommon and have no necessary connection with the sequelent *broncho-interstitial pneumonia* in the *corn-stalk disease*, any more than with the lung plague. I shall describe and illustrate this lesion as it occurs in the corn-stalk disease, in a way that will show that such novices as examined these cattle would indeed be strongly warranted in suspecting the contagious lung plague, and yet it was not, and no experienced eye would ever think it was. How many hundred such cases the British inspectors may have seen since 1879 I do not know, yet if it is on such grounds that they have "damned" our cattle, then may a more merciful judge have pity on them. Among these men we find the names of McFadyean, a man who pretends to edit a *Journal of Comparative Pathology* and to be a bacteriologist, Professors M'Call and Walley, presidents of two of Britain's veterinary colleges, and Brown, now president of another and for years and still the veterinary adviser of the privy council of England. But one name we do not find in the condemners; it is that of Prof. William Williams, of the New Veterinary College of Edinburgh, a man who for years has rightly enjoyed the reputation of being perhaps the only pathologist among the veterinarians of England; an honest worker and clear-headed Welshman.

This brings us to another factor that has been mentioned as playing a part in the condemnation of our cattle:

Second—*Jealousy of one man.*

There is no question but that the veterinary profession of the world, at least that outside of Britain, has given to Prof. Williams the reputation that I have attributed to him, and his various writings fully justify it. In Britain the same unfortunate conditions exist in regard to medical and veterinary colleges as in this country. They are all either endowed or private incorporated educational institutions. They are all business ventures, dependent on students' fees for their successful existence. President Williams is, and has been, a remarkably successful man. He stepped out of an old school and started a new and rival one by his own energy and means. He has for years been an ambitious and disturbing element in British veterinary matters and generally obtained the advantage of his opponents. On such questions as this, when opposed to others, time has generally justified his conclusions. The discussions which have taken place for years on this very question have ever betrayed the jealous animosity of his opponents. They are the heads of, or connected with, rival schools. "To be downed," as we say, would of necessity injure their schools. Hence, the rivalry. Hence, I think it is not going too far to assert that professional jealousy has led men, otherwise worthy as such, to adhere to opinions once expressed even when, as now, every iota of evidence shows them to have been in the wrong.

This rivalry between experts leads to exactly the same results in this country and is taken advantage of in forensic cases by lawyers on both sides. On a question in which there is no chance for dispute and all educated medical men agree, I have seen "professors" from different medical schools, whom I knew personally, privately agreed perfectly, perjure themselves and contradict one another simply because of this opposition and that the fact might advertise them and their schools by being noticed in the daily papers. This is "medical science" as displayed by many school men of to-day.

We now come to the last factor mentioned:

Third—*To keep their jobs, or Britishism vs. Americanism.*

That it is natural to men to desire to retain their positions or to obtain government employment, "to keep solid with the authorities," is common to the majority of men under every condition and in every country. We cannot blame them for this. It is human, as is the more absurd idea of "standing by one's country whether right or wrong." That an intense commercial rivalry, much embittered by our inhumane protective tariff, exists between Britain and this country is true. It may be natural, but nevertheless it is inhumane. Nationalism must eventually give way to the more humane internationalism. The most strong patriotism should ever be tempered with justice. The Britains should not be blamed in this matter. Why should the "pot call the kettle black"?

It has been said that Prof. Williams has been invariably opposed to the assertion of his contemporaries, that the disease called by them "*contagious pleuro-pneumonia in American cattle*" was in reality the lung plague. Let us hear what he has to say on the question, quoting from *North British Agriculturalist* of February 17, 1892:

> "I have in the first place to give you a short history of the controversy which has arisen upon the subject of my illustrations. And as there are other matters of business on the agenda of the meeting, I will be very brief.
>
> "Early in 1879 pleuro-pneumonia was reputed to have been found amongst American cattle landed at Liverpool, and the shippers there, in order to satisfy themselves of the correctness of the diagnosis of the veterinary advisers of the privy council, and who now hold the same position on the Board of Agriculture, requested Professors M'Call, Walley, and myself to visit Liverpool and examine the cattle, and watch the *post-mortem* results; and here, gentlemen, commenced the dispute as, after having seen a few diseased lungs, I maintained that the malady was not pleuro-pneumonia but a catarrhal or broncho pneumonia, combined in a few cases with pleurisy, but in others the pleurisy was absent. Professors M'Call and Walley agreed with the advisers of the privy council, and I found myself in a magnificent minority. During the spring and summer of that year several

condemned lungs were sent to me from Liverpool, and after further investigation I found no reason to change my opinion.

"The matter remained in abeyance so far as the profession was concerned until April, 1891. It is very true we now and then heard that pleuro had been detected amongst American cattle landed in this country, but we had no means of verifying or contradicting these reports; but some two years or so ago the American government sent inspectors to the various ports where American cattle are landed for the purpose of investigating and reporting upon the cattle slaughtered; and, wonderful to relate, we heard no more of American pleuro, except one notable report from Dundee, until a lung of an ox was found at Deptford in April, 1891, and said by Professors Brown and Duguid and Mr. Cope, and confirmed by Professors Walley and M'Fadyean, to have *pleuro-pneumonia contagiosa,* and in further support of the correctness of this diagnosis, Mr. Chaplin, the minister of agriculture, said it was pleuro, *for he had seen the lungs.* But Dr. Wray, the American veterinary inspector at Deptford, disputed this opinion, and on 18th April brought a portion of the lungs to me.

"I examined this portion of lung along with my son, and we both came to the conclusion that it was not pleuro-pneumonia. I was again in the minority, and nothing more would have been heard of the matter, at least so far as I am concerned, had not a remarkable coincidence occurred in Paris during the winter of last year, 'when it appears that MM. Redon, Godbille, and Blier, well known veterinarians and sanitary inspectors at La Villette—the Parisan cattle market—where thousands of American were exposed for sale. On three several occasions these veterinarians affirmed that some of the animals were affected with an unusual disease of the respiratory organs. In November, 1890, in a lot of more than 400 cattle direct from Illinois and Indiana, one died and two were very ill, the symptoms leading to the suspicion that they were affected with contagious pleuro-pneumonia.

"'The sick animals were killed and the lungs examined by Godbille, who found no pleurisy, but such a suspicious appearance that, considering the importance of the matter, the two sets of lungs were sent to Alfort, to be submitted to M. Nocard, one of the most illustrious of veterinarians and pathologists, and he reported that at the first glance a section of the hepatized tissue presented the appearance of a recent lesion of *pleuro-pneumonia contagiosa.* The tissue was dense, compact, friable, the color varied from bright red and deep brown to almost black, whilst the lobules were isolated from one another by thick septa, infiltrated with a considerable quantity of yellow limpid fluid, etc.'—See *Veterinary Journal,* Oct., 1891.

"Now, gentlemen, these appearances were startling, and if the investigator had been a smaller man than he is, he might have made his conclusions from these naked eye apearances, and said it was contagious pleuro; but there were great international and commercial considerations in question, as well as the scientific bearing of the subject, and Nocard did what our own government officials ought to have done, if not in 1879, at least in 1891. He made further investigations, and arrived at the conclusion that he had other than pleuro to deal with. He found in the first place that the infiltration of the connective tissue was less abundant, the serum less albuminous; that the tissue of the lobule in the thickened girdle of connective tissue had not the uniformity of tint and consistence that characterize pleuro-pneumonia lesion; that it was harder, more manifestly hepatized in the center than at its periphery—the lesion extended from the *bronchial and not from the perilobular tissue.* In 'pleuro,' it is the opposite, notwithstanding what Drs. Yeo and Woodhead have stated, and which statements have been partly accepted by Professor M'Fadyean.

"There was another important differential sign. Pressure caused a notable quantity of thick viscid light yellow pus—analogous to that observed in certain forms of verminous broncho-pneumonia—to issue from the bronchioles (this condition was very manifest in the Liverpool cattle in 1879); but a micoscopic examination of this fluid and of the lung-pulp did not lead to the detection of ova, embryos, or worms of any kind. The bronchial mucous membrane was inflamed, thickened, corrugated, and more or less denuded of its epithelium, while the submucous connective tissue was infiltrated with yellow serum, and considerably thickened in places.

"This muco-pus from the bronchioles was found to contain an abundance of short, oval, mobile bacteria, which appeared to be the only microbes present. The organisms were also found, as if in a state of pure culture, in the hepatized tissue, and more especially in the limpid serosity that distended the perilobular lymphatic sac. This single character alone sufficed to affirm that the lesion was not of a pleuro-pneumonic kind; for it is well known that the lung serum in that disease is very poor in figured elements, and that when it is collected pure from the infiltrated septa, it does not usually contain any microbes.

"In this instance the microbe, which existed in such numbers, belonged to the large class of ovoid bacteria, of which the rounded poles, fixed aniline colouring matters more strongly than the centre. It is well known, as pointed out by Nocard, that the fowl cholera, the duck cholera, the septicæmia of rabbits and ferrets, the game plague, the swine plague, etc., all have analogous microbes, and which can only be distinguished from each other by the collective biological characters, and more especially by the effects of their inoculation on different kinds of animals.

"Experimental inoculations were now made by M. Nocard, when he found that mice, rabbits, guinea-pigs, and pigeons, when inoculated subcutaneously with two or three drops of the serum or the culture, succumbed in less than forty-eight hours, without œdema at the point of inoculation, with intense congestion of all the viscera, but without any definite localization.

"Sheep and calves inoculated subcutaneously, or in the trachea, with a cubic centimeter of culture, of serosity, or of virulent pus, did not die; they suffered however from intense fever, and remained prostrate, without appetite, for some days, but soon regained their normal condition.

"Intra-pulmonary inoculation was more effective, as a calf of eight months and a ram of two years, inoculated in the right lung with five drops of peritoneal pus from a guinea-pig, died in less than forty-eight hours with fibrinous pleurisy and exudative broncho-pneumonia analogous to that observed in American cattle, the lesions being very rich in bacteria. Nocard concludes that the disease does not resemble any known in France, and considers it is a malady special to American cattle, and is inclined to think that it is the affection known in the western states as the 'corn-stalk disease,' described by Billings, of whose observations there is an account by Bowhill in the last number of the *Veterinary Journal.* And the question arose as to whether the importation of this microbic broncho-pneumonia would prove a source of danger to France. M. Nocard was able, however, from his close observation of facts, and the experimental study of the conditions of contagion, to state that the malady had only very feeble contagious properties. In three very large importations in which the disease was detected, notwithstanding the considerable number of animals in each, and their long and close contact with each other, the affection did not spread, and the cases remained isolated ones. From all these facts, his opinion was there was no urgent danger, or any necessity for special measures.

"Again, Professor M'Fadyean stated in Liverpool that Mr. Bowhill, who, he observed, had served a short apprenticeship, had shown a microbe, and sneeringly described it as an ordinary putrefactive one. This was, to say the least of it, very unfair and very unprofessional, as Mr. M'Fadyean never saw it. If Mr. Bowhill is only an apprentice, it must follow that Nocard, Billings, Dr. Hunter, and myself are also apprentices, as we have all seen the same microbe, and both Billings and Nocard have proved the pathogenic importance of it by inoculations; but in order to prove the fallacy of Mr. M'Fadyean's uncalled-for remark, I wrote to Dr. Wray, and I append his reply, with the simple observation that when the lung arrived in Edinburgh it was free from putrefaction, and is so to this day, as you can easily verify in examining the part before you.

"Now, in conclusion, I have simply to say that the American disease is not pleuro, but that it agrees in every respect with what Billings and Nocard have described as broncho-pneumonia, and that it is identical—but the specimens show a more chronically-diseased condition—with what I discovered in both States and Canadian cattle landed in Liverpool in 1879.

"One other observation. I trust that this controversy will lead to good results; that veterinary surgeons in future will have greater faith in microscopic investigations, as by such methods it has been proved that pleuro-pneumonia is a disease commencing in the perilobular tissue, and that catarrhal or broncho-pneumonia proceeds from the tubes and air cells, from which pus can be pressed out, and that its best result is seen to be a desquamation of the epithelium of the inflamed tubes; that in pleuro fibrin is exuded in abundance into the connective tissue, air vesicles, bronchioles, and blood vessels, where it coagulates into permanent clots; that pus is seldom or never found, and that in broncho-pheumonia this remarkable condition is absent."

It may be well, also, to read what some other British veterinary writers have said on the same subject, in order that we may well understand the position that has been taken in England. For this purpose some editorials from the *Veterinary Journal*, of England, will be now quoted:

"*The Existence of Contagious Pleuro-Pneumonia in the United States*

"That the contagious pleuro-pneumonia of cattle exists in the United States is evident from the condition of some of the animals imported into this country from across the Atlantic, from the reports of the professional journals published in that country, and also from the official report issued by the United States Department of Agriculture. That the malady is also prevalent in a number of centers is no less evident from the information derived from these sources. An attempt to introduce live stock, unless for immediate slaughter, from any country in which this most serious of all animal scourges exist, should be resisted at all hazards at the present time, when we are making such strenuous efforts, at such heavy cost, to free these islands from it, and our determination to keep out fresh installments of infection cannot surely be regarded as anything but laudable by other countries, no matter though their cattle traffic may be impeded thereby. It would be nothing less than the height of folly, and we have already suffered terribly through our folly, stupidity, and supineness in dealing with the scourge, to allow store cattle to come among our herds from regions in which 'pleuro' exists, even though assured they were not drawn from infected districts. We are only too painfully aware of the value of such assurances through our dealings with Ireland, and the cattle trade is pretty much the same all over the world. Inspection at the port of embarkation, and even at the port of debarkation, is of very little if any value when we consider how occult the malady is, and that an animal apparently in good health may yet be so diseased as to be a bearer and active disseminator of infection. No contagious malady of animals is so difficult to detect or diagnose, and though the affection may be in the lungs *perdus*, as far as human agency can discern, yet for months it may be transmissible in this condition, and taint every healthy bovine brought near it. It must, therefore, be a long time before a country in which the malady prevails in various centers and in which there is movement of stock, can be declared free, even under the most severe and active sanitary measures, such as we are now trying to enforce. It is to be hoped, therefore, that no cattle from infected countries shall now, or for the future, be allowed to go beyond our port abattoirs until these countries have been, on reliable authority, declared entirely free from the malady for twelve months. If their cattle trade with the United States is worth maintaining, this will be a strong inducement for them to exert themselves in securing a clean bill of health as quickly as possible. Traffic in cattle may be beneficial to nations; traffic in contagion cannot be." *Veterinary Journal*, London, Eng., 1891, Vol. 1, p. 253.

From my school days in Berlin, and from a general acquaintance with veterinarians of note, a skeptical and cynical character, as far as I am concerned, regarding their pathological knowledge and actual experience I think I can see the reason, or perhaps better, a reason why the English inspectors might have made the mistake they undoubtedly did, most probably universally as regards our cattle; the tendency to this mistake being undoubtedly increased by the professional and other influences which have been alluded to.

All organs are anatomically and physiologically made up of two great classes of tissues, and when we classify pathological tissues in them the first great differentiation is made on this basis. These two classes of tissues are known as the *parenchymatous*, or functional active tissues, and the interstitial, or supporting tissues, the frame-work of the organ. Inflammatory processes are then classed as parenchymatous or interstitial, according to the tissues primarily and most severely complicated. This is as applicable to the lungs as any other organ, and hence we have parenchymatous and interstitial pneumonias, the first being by far the most common variety. In fact, the only example of a widely dispersed interstitial pneumonia to which our attention has been called, both as students and practitioners, has generally been the *contagious pleuro-pneumonia* of cattle, and I think it can with truth be said to-day that it still remains the only example of a specific and general interstitial pneumonia in any species of animal life. Until very lately we did not know that any process in the lungs of cattle could so closely resemble that disease as can this sequelent *broncho-interstitial pneumonia* in the *corn-stalk disease*, for it is not proper or necessary to that disease, which is a specific septicæmia, but a result in cases of longer duration than investigators would generally come across, or, in fact, have called to their attention in this country. It is only since November last, and again very recently, that I have seen a piece of lung in this disease of the kind so closely resembling *contagious pleuro-pneumonia* as to lead to a momentary suspicion of the existence of that plague. I have seen an immense number of such lesions of a circumscribed broncho-pneumonic type, such as our government veterinarian says were condemned as belonging to the "contagious" plague by the English inspectors when he was in that country in 1880, a few illustrations of which have been quoted. Another fact, apt to be misleading, which has already been alluded to, is this, the lung tissue in the immediate vicinity of these chronic broncho-pneumonic centers in cattle lungs must of necessity generally be somewhat irritated and congested, if not pneumonic, and it is very seldom that even a circumscribed condition of this kind can occur in cattle lungs without more or less œdematous swelling of the interstitial tissue in the complicated parts. Many cattle, otherwise perfectly healthy, come to the slaughter bench with such a circumscribed broncho-pneumonic lesion in the lungs, often very small and sometimes but a single one. They are frequently the only indication left that as calves such individuals have had lung worms and otherwise fully recovered. Mr. Plimsoll, of England, deserves the greatest credit for his humane spirit in calling attention to the abuses cattle suffer on the transatlantic steamships, more especially such as are shipped in the "between decks." The conditions of close air and no movement there abounding must of necessity lead to the aggravation and extension of any such local lesions in the lungs of cattle, and such aggravation and extension would add the lesions of acute pneumonia and increase the interstitial complications in lungs having such weak centers; but even with all this there is no reason that such serious errors in diagnosis should have been made and continued for such a long term of years with no scientific investigation on the part of the governments of Britain and the United States. It is true that this govenment continually protested against the condemnation of cattle and the "bad" name given our whole cattle breeding industry; but these protests were not supported by such evidence as must of necessity be conclusive of their value.

Notwithstanding the examinations and conclusions made by our representatives in 1880, and the positive assertions of Prof. Williams, our chief argument has been *"that there was not and never had been any contagious pleuro-pneumonia in the cattle of the western states."* Our skirts were not free from all suspicion, however. For about thirty years we had to admit that we constantly had the lung plague in our cattle in some parts of our seaboard states, and, as our own government reports show, only too extensively. Hence, that kind of argument was next to valueless. The British authorities could equally well ask (especially in the face of the fact that contagious pleuro-pneumonia had escaped the barriers of the east and broken out quite severely in Chicago in 1886–87, and no one knows how many years it had been there to any exactness): *How do you know it is not scattered in your western herds? What guarantee can you give us that it is not there?*

To say it had "never been seen in western cattle" is no answer to such a question, considering the generally chronic nature of the malady and its difficult diagnosis in isolated cases, and the almost total lack of any well organized veterinary police in this country.

To be sure, the immense slaughtering of beeves at our western cattle marts could be quoted in evidence, and we could with tolerable certainty assert that *"no case suspicious of lung plague had ever been seen,"* but that assertion and such experience certainly was not strong enough evidence to remove a doubt which the British government is naturally inclined to strengthen all it can.

Notwithstanding even the results of our Nebraska investigations and the very conclusive testimony of Nocard and Bowhill in support of the same, still our skirts are not altogether clear of a lingering suspicion and cannot be until every trace of contagious pleuro-pneumonia has been removed from this country.

It will take drastic measures to do this, but it can be done. *Law or no law it must be done.* There is law enough somewhere by which it can be done. Congress should stop its nonsense and make a sufficient appropriation to insure its being done, even if it "busts the bank." I am very well aware that one great obstacle lies in the indifference of the local authorities in the states where the disease exists and the uncertainty of national authorities in such matters; but the interstate commerce law can be used and not a bovine allowed to cross the borders of an affected state for any cause whatsoever, no matter how unsuspicious it may be. Were I the President or secretary of agriculture I would do this if it took the United States army to control the traffic. Not a cattle car should be allowed to leave such states unless scalded, scrubbed, and whitewashed. The people of two or three states might "kick," but those of the great cattle-growing states would soon teach even mighty and corrupt New York that she was a very "small toad in the puddle" after all. *A more practical and less disturbing, and eventually less expensive, method, which would equally well receive the support of nearly the whole country, would be to at once buy all the cattle at all open to suspicion and make a holocaust of them, barn-yards, stables, and all, and build the owners new buildings and pay for all the cattle.* Though I have no love for the present secretary of agriculture, still I know enough of him to know that he is equal to just such an undertaking if congress will supply the means and authorize him to "go ahead." It is very sure there would not be a suspicious bovine "head" in the country by next January *if congress would only do its duty.*

That the British government has not been and does not mean to be so very unfair in this matter is shown by the very manly action of its allowing this government to send its inspectors over there to examine the slaughtered cattle for themselves and for us. This manly concession seems to have been rather surprising to the British veterinarians, if we can judge anything by the following editorial:

"*American Veterinary Inspectors at Our Ports.*

"It is quite a new departure to allow a foreign government to send inspectors to our ports to examine imported cattle, as if our own inspectors were not to be trusted to decide fairly as to whether animals landed are affected with contagious diseases or not. American authorities profess disbelief in the statements of our veterinary officers as to the detection of contagious pleuro-pneumonia among cargoes of cattle received during the present year from the United States, and so they are allowed to send inspectors to the ports of London, Liverpool, and Glasgow in order to satisfy their doubts on the question. The policy of allowing this privilege is of doubtful expediency, to say the least of it. As the American officers may come to us with the foregone conclusion that the disease complained of is only sporadic pleuro-pneumonia and disputes between them and our inspectors must be the inevitable result." *Veterinary Journal*, 1890, Vol. XXXI, p. 180.

The results of this inspection and that of 1880 by American veterinarians, confirming, as they do, Prof. Williams' repeated assertions that the disease in our cattle over there is not *contagious pleuro-pneumonia*, which opinion is most strongly supported by a more exact scientific examination of lung tissues, of a condemned bullock that all the British inspectors most positively asserted did have *contagious pleuro-pneumonia*, certainly either does not speak well for their ability as "experts" and supports the suspicion that the factors, previously considered, still bias their judgment.

It is somewhat singular, that after having continually condemned our cattle ever since 1879, the first really elaborate study of the lung tissues of such a condemned animal, in Britain, should have been made by a student of my own, and an unusually gifted man, especially in pathology, without which no man can ever become a reliable patho-bacteriological investigator. I allude to Dr. Thomas Bowhill, who spent about six months with me in the fall and winter of 1886–87 and who, I am happy to say, is to again become a resident of this country having taken to himself an American "rib" to support his not over-tottering frame. In the *Veterinary Journal*, London, for February and March, 1892, Dr. Bowhill published an article in which he clearly demonstrated that the "Deptford" steer was afflicted with the sequelent *broncho-interstitial pneumonia of the corn-stalk disease. I have myself examined some of his slides, compared them with my own work, and most absolutely confirm his diagnosis*, though we may both be mere amateurs in comparison to such magnificent specimens of dilettanteism as McFadyean, Walley, and some other British veterinary pathologists. The following applicable quotations are from Dr. Bowhill's paper, the balance being quotations from my own work, and have reference to his personal studies of the "Deptford" lung:

"We will now consider the points of identification (or rather the evidence in support of the same) between the '*corn-stalk disease*' and the so-called *pleuro-pneumonia contagiosa* in an American ox landed at Deptford. My remarks are limited to the conditions present in a piece of the lung of that animal forwarded to me by Prof. Williams, who at the same time sent the piece of a lung of an animal affected with undoubted *contagious pleuro-pneumonia.* Microscopical examination of a section of the Deptford ox's lung reveals many points of identity with the lesions mentioned by Dr. Billings as occurring in the "corn-stalk disease."

"First—The condition of the bronchi is very striking, especially the swollen condition of the mucosa and infiltration of the submucous connective tissue. (See Nocard.)

"Second—The plugged vessels with grayish, dense-packed material described by Dr. Billings is also present in the Deptford lung, as well as the thread-like lines (also described by Dr. Billings) running through the dense material plugging them. (This condition might lead a careless or inexperienced observer to mistake the lesions for those of contagious pleuro-pneumonia.)

"Third—The dense-packed condition of some of the alveoli, described by Dr. Billings, as well as the round-cell infiltration in the interstitial spaces, are both well-marked lesions in the Deptford lung.

"Fourth—We have to consider, or to account for, the presence of bacteria in sections of the Deptford ox's lung, morphologically identical with those described by Billings as the cause of the 'corn-stalk disease,' and by M. Nocard in infectious broncho-pneumonia in American cattle landed in France.

"The piece of lung having been in alcoholic cultivation, experiments were impossible. At the same time that sections of this lung were stained for histological purposes some were also stained for bacteria. I noted the presence of ovoid bacteria in sections of the Deptford ox's lung; they fixed the analine color most intensely at their pole ends and measured one micro-millimeter long and three-fourths to four-tenths broad, and furthermore, they could be differentiated in the lymph-spaces resembling a state of pure culture. Nocard says: *This single character alone sufficed to affirm that the lesion was not of a pleuro-pneumonia kind.* They could not be stained properly by the Gramm method, as these bacteria are decolorized on contact with iodine."

Although Prof. Williams has extensively quoted from Nocard's publications, still, as it bears such direct relation to the case in point and particularly on Bowhill's investigations, it is deemed advisable to place a translation of it in more detail in this place. It is only necessary to add that no greater authority, as a pathologist, honors the veterinary profession of any country to-day.

"Infectious Broncho-Pneumonia in American Cattle" (Corn-stalk Disease). By M. Nocard, Professor of Veterinary Pathology, Alfort, France.

"During last autumn several thousand magnificent American cattle were placed on the market at La Villette. The cattle were from Indiana and Illinois. At three different examinations the sanitary officials observed in some of these animals a disease of the lungs different from any known in France. On the 15th of November, 1890, attention was called to a very sick animal among these American cattle. On necroscopic examination it was found that the anterior lobe of the right lung was the seat of recent and acute consolidation; the absence of complications of the pleura, the slight serous infiltration of the interstitial tissue, spoke very strongly in favor of a simple pneumonia; nevertheless the inspector did not feel assured that it was not an abnormal form of contagious pleuro-pneumonia.

"On the morning of the 16th of November it was found, on inspection of the American cattle, that one had died during the night, and that others presented phenomena suspicious of contagious pleuro-pneumonia.

"The commissioners consented to the immediate slaughter of the two animals. The autopsy demonstrated lesions in the lungs identical with those observed in the previous animal. Before making a decison in question of such grave importance the lung of the two animals were brought to me by M. Redon in order to obtain my view of the nature of the lesions.

"At first sight the cut surface of the consolidated tissue presents the appearance of the lesions of a fresh case of contagious pleuro-pneumonia; the tissues of the diseased portions of the lungs are dense, firm, compact, friable, varying in color from clear red to a brown or even blackish shade; the lobules are isolated one from another by thick, interstital tissue infiltrated by a considerable quantity of yellowish, limpid fluid. More exact examination demonstrated notable differences between these lesions and those of contagious pleuro-pneumonia. The serous infiltration is less abundant, the fluid being less albuminous and of a less yellow color, having a more limpid character; pressure showed the lymphatic sacs to be distended to excess by small concoctions of a white, firm, fibrinous character. The intra-lobular tissue is increased to solidification, not having the uniformity in consistency or color which characterizes the lesions of contagious pleuro-pneumonia; it is more hard, more manifestly solidified in the central than peripheral portions; *the lesion proceeds from the bronchial tubes and not from that embracing the lobuli;* the contrary is the case in contagious pleuro-pneumonia; where the lesions are of very recent origin one often sees the intra-lobular tissue dense and blackish at the periphery, whereas the center of the lobulus is still red, soft, and permeable.

"Another lesion of differential importance is, that pressure on the walls of the bronchioles causes the exudation of a notable quantity of mucus matter, thick, viscid, yellowish-white in color, much resembling that observed in certain forms of verminous broncho-pneumonia; microscopic examination eliminated that hypothesis. On opening the complicated bronchial tubes the mucosa was found inflamed, thickened, and more or less desquamated from its epithelium; the submucous connective tissue was infiltrated with a yellowish fluid, and was sometimes considerably thickened. This mucus matter from the bronchial tubes contained short ovoid movable germs to the exclusion of all other microbes; they were present in a veritably pure culture in the hepatized tissue and above all in the clear serous fluid which distended the perilobular lymph-sacs. This single characteristic sufficed to show that the lesion had no relation to contagious pleuro-pneumonia, the serous effusions in that disease being characterized by their extreme penurity in morphological elements, scarcely ever leaving any germ forms. This microbe, which existed so abundantly in the pulmonary lesions, belongs to the first class of ovoid germs whereof the pole-ends color much more markedly than the central portion of the body, such as those of hen cholera, the cholera of geese, rabbit septicæmia, the ferret septicæmia, the Wild-seuche, and swine plague.

"This, the latest discovery in the series, is very small, is motile, colors well in the usual solutions, the central part of its body remains clear after tinction in fuchsin or methylin-blue; it does not color by Gramm's method. This germ is easily cultivated in the different media usually used, and is both an- and aerobic; the reaction of the cultivating fluid is not sensibly modified; it does not coagulate milk; it preserves its vitality and virulence a long time. Inoculated delicately on oblique peptone gelatine, the germ does not develop very abundantly, the growth being a thin, somewhat bluish coating, translucid, with festered edges; does not liquefy gelatine. Mice, rabbits, guinea-pigs, and pigeons succumbed within twenty-four hours to subcutaneous inoculation of two or three drops. Sheep or cattle inoculated subcutaneously or into the trachea did not die, but had an intense fever, loss of appetite for several hours, but finally recovered. Intra-pulmonary inoculation is more efficacious; small quantities killing in forty-eight hours, the animals presenting a fibrinous pleuritis and exudative broncho-pneumonia analogous to that seen in the American cattle. These lesions were extremely rich in bacteria. *A calf and two sheep that has survived subcutaneous inoculation and in the trachea resisted an intra-pulmonary inoculation of ten drops of a virulent culture later on,* which may indicate a non-recurrent disease, and promises much for the future.

"The hen, the dog, and the rat resisted inoculation. This disease appears to be probably a special one of American cattle, and while unknown in France is known to American veterinarians. I have made some investigation in this connection, and discovered that it is known in the west under the name of the 'corn-stalk disease,' literally '*Maladie du Chaume de blé.*' The report of Billings, from Nebraska, 1888, corresponds with sufficient exactness to my own observations." *Recueil de Méd. Véterinaire*, Vol. VIII, 1891, p. 424.

SHOWING HOW A LITTLE SCIENTIFIC LEAVEN WORKED IN BRITAIN.

Two editorials from the London *Veterinary Journal* have previously been quoted to illustrate the opinion of the profession there on this question of *contagious pluero-pneumonia* in American cattle. The publication of the above quoted article by M. Nocard led to the following decided modification of the editor's opinion. An Englishman dies hard but is generally a royally game fellow.

"*Contagious Pleuro-Pneumonia and Infectious Broncho-Pneumonia in Bovines.*

"It is now some years since a diversity of opinion occurred with regard to the presence of contagious pleuro-pneumonia in a cargo of American cattle disembarked at Liverpool, and which were condemned because one of them was found to be affected, according to the government authorities, with that disease, but which, in the opinion of Prof. Williams after inspecting the lung lesions, was not that malady but another. Quite recently a similar case occurred among another cargo of American cattle, and the same veterinary authorities stood in the same position in regard to its nature; this time Prof. Williams being supported by the United States veterinary inspector, who has been sent to this country to see that the cattle imported from the states are not unfairly condemned. This course was deemed necessary from the fact that many cargoes from that country have been accused of being infected with the lung plague which the veterinarians on the other side have declared could not be. The recent case, just referred to, has caused a more than usual amount of interest, from the striking difference in opinion as to the nature of the changes in the lungs of the diseased animals; in fact, as to whether these alterations were those of lung plague or of another form of pneumonia. Not long ago we were shown photo-micrographs of *pleuro-pneumonia contagiosa* and this American cattle disease, which Prof. Williams has designated *broncho-pneumonia catarrhalis*, and certainly the difference between the two is very marked.

"This divergency of opinion as to the nature of the malady for which ship-loads of American cattle have been condemned has had no result other than the opinion of the English government experts has

prevailed; and nothing further has been done to settle the matter, though astonishment may be expressed that experimental proof, so easily obtained, was not resorted to."

Strange to say, corroborative evidence in favor of Williams' contention has sprung up in a very unexpected quarter, and on the statement of one of the foremost veterinary pathologists in Europe, who probably knew nothing of what was taking place in England in reference to this matter.

(Were it not for Nocard's investigations we should probably still be told of a new and alarming "contagious disease" in American cattle, had it not been for our investigations in Nebraska, which it seems the Britishers have never read, though their colleges and the veterinary department of their privy council have been favored with complimentary copies. Our Agricultural Department at Washington had also to be taught by a foreigner that there was after all such a thing as the "corn-stalk disease," the "dry murrain" of their own brains having prevented them from coming to a realization of that fact before, though our report has now been published three years.)

The illustrious editor of the London *Veterinary Journal*, after giving the results of Nocard's investigations, resumes upon them as follows:

"Nocard arrives at the conclusion that the disease does not resemble any known in France, and he considers it as a malady special to American cattle; that it is not rare is proved by the fact that since the month of November (1890) he has seen it, always presenting the same features, in other lots of cattle from the United States—Indiana and and Illinois. He is inclined to think that it is the affection known in the western states as the 'corn-stalk' disease, but however this may be, it results from his observations and investigations that American cattle are liable to a microbic broncho-pneumonia hitherto unknown in France, and the serious question therefore arose as to whether any danger was to be apprehended from the importation of such animals. From his observations, Nocard expressed the opinion *that there was no urgent danger nor any necessity for special measures.*

"The question that now arises on this side of the channel is one of some moment, not only from an economical, but also from a pathological point of view. *Many cargoes of American cattle have been condemned because it was found that one or two in each lot were affected with what was supposed to be the specific and only too well known lung plague.* United States veterinarians have strenuously denied the ex-

istence of that malady among the cattle, and Prof. Williams, when the matter was referred to him on two occasions, has concluded that the lung plague was not present, but that the lesions were those of catarrhal broncho-pneumonia, and now Nocard finds what we are almost forced to conclude is the same disorder, examines it and experiments with it as a scientist of his position and responsibility should do, and, *mirabile dictu*, arrives at the same conclusion as Williams, and even designates it by the same name. *It will be an extraordinary revelation if what has been considered as contagious pleuro-pneumonia in American cattle should turn out to be something very different and of little, if any, importance.* The slaughter mania is a terrible barrier to pathological observation, and little as we have done in the way of scientific investigation of animal diseases in this country, it is to be hoped that the example of France in this instance will not be without effect. It is quite possible that the American cattle found in La Villette market would have been condemned here as infected with contagious pleuro-pneumonia, and the usual consequences would inevitably follow. Must we again say that in this instance, as in so many others, "*they manage these affairs better in France*"? *Veterinary Journal*, Vol. XXXIII, October, 1891, p. 260.

WERE THERE OTHER FORMS OF INTERSTITIAL PNEUMONIA IN CATTLE THAN THE CONTAGIOUS KNOWN TO VETERINARIANS PREVIOUS TO THESE LATTER DAY INVESTIGATIONS?

The fact has already been alluded to, in partial excuse for the mistakes of the British veterinary inspectors, that the general opinion instilled into the minds of veterinary students, and hence, becoming part and parcel of their post-graduate knowledge, has been, and today largely is, *that no other form of interstitial pneumonia, giving to the lungs of cattle anything resembling the "marbled" appearance of the contagious lung plague, ever occurs.*

We know better now, though I am still convinced that we have no disease in cattle which gives to the lungs that distinct, sharply outlined, "marbled" appearance which is now, and ever will be, "*characteristic*" of contagious pleuro-pneumonia. *The condition met with in the lungs of cattle resulting on the corn-stalk disease, while often a chronic interstitial disturbance and somewhat resembling the lungs in contagious pleuro-pneumonia, is so markedly different and generally so washy and broken in outline, in comparison, that any competent veterinary pathologist should be able to recognize it at a glance.* That it can be done is shown by the verdict of Williams and Nocard. In

alcoholic specimens the case might be more difficult, but if the specimen comprises a considerable extent of lung there should be but little difficulty even then. No excuse can really be offered for mistaking circumscribed centers of chronic broncho-pneumonia, as has been done so often by the British veterinary inspectors for the lesions of *contagious pleuro-pneumonia,* nor for the sequelent *broncho-interstitial pneumonia* of the *corn-stalk disease,* which should never be considered specific to that malady.

In order to show that I am right in what has been said regarding our teachings in the past as to the specific and limited character of chronic interstitial processes to the lung plague is supported by the following from my most honored and loved departed friend, Gerlach, director of the Berlin veterinary school during my studies. No one will accuse me of partiality when I say that no veterinarian of his day was considered to be a greater authority. His work on "Gerichtliche Thierheilkunde" was "standard" then and has not yet ceased to be so in many respects. I heard his lectures on this very point and have since had to unlearn his instruction as the result of very extended personal observation. I dare affirm *that no chronic inflammatory process can exist in the lungs of bovines and be seated in the bronchial tubes, which must not of necessity extend somewhat to the immediately connected interstitial tissue and adjoining parenchyma; the disturbance may be very circumscribed that will not produce a more or less extended induration of the interstitial tissue which, taken by itself and without consideration of the conditions in toto, will present a somewhat "marbled" appearance of that portion of the lung. The anatomical structure commands that this be so.* No one should mistake such a condition for that of contagious pleuro-pneumonia, however.

Gerlach emphatically denied the position I have taken in the above statement, though persistently opposed by such men as Haubner, and other of his contemporaries, men of almost equal eminence. The views of Gerlach were those prevailing at the time and as the past is always too much of the present, they still rule, though decidedly mistaken. He says:

> "From the anatomical point of view, the assertion has previously been made that the "marbleization" of the hepatized lung is chiefly due to an anatomical (structural) cause, and, therefore, in every hepatization in cattle lungs must be characterized by a marbled appear-

ance [That statement of Gerlach's is too general; his opponents did not say "in every hepatization," but that under other circumstances than those of contagious pleuro-pneumonia a "marbled" condition could and did occur.—B.], *and that therefore the marbled appearance of the cut surface is not characteristic of contagious pleuro-pneumonia.* This assertion is but in part right, that is, *in so far* as the marbled appearance is made possible by the anatomical structure; decidedly false, however, is the further assertion, that on account of the anatomical structure every hepatization in the bovine lung must be marbled. To produce that condition something else is necessary, something specific."

The specific in this case is—

First—The anatomical structure.

Second—The widely-dispersed irritant (the cause) in the lungs.

Third—That this cause exerts its primary and (this is specific) evenly-distributed irritation in and from the lymphatic system of the interstitial tissue, producing an evenness in the complicating induration not seen in any other disease.

Fourth—That the specific irritans exerts a chronic action. All three of these conditions can be met in a varying degree, but never generally, or evenly, by some other cause, than the irritans of contagious pleuro-pneumonia.

Again Gerlach says:

"Aside from contagious pleuro-pneumonia in cattle, I know of only a metastatic, or a foreign body, and a tuberculous pneumonia, and in all of these a marbled condition never occurs. *I can and must assert that this marbled condition in the bovine lung is pathognomonic for the lung plague.*" (P. 414.)

Gerlach was right as well as wrong. Neither he nor his opponents seem to have understood one another, for I have read them both. Gerlach meant what I have stated in clause No. 3, above, *the general and even distribution of the marbled appearance, with varied parenchymatous complications,* Haubner and others meant, and rightly, that marbled conditions strongly resembling the same did occur from other causes than the contagious lung plague.

This shows that conditions so strongly resembling those of contagious pleuro-pneumonia as to have excited the attention of veterinarians and caused discussion were known as far back as 1872, and hence, that even the British inspectors must be blamed for not having a knowledge of these things.

"Septic Pleuro-Pneumonia in Calves."[*]

This disease seems to be quite common in Holland, and as the etiological micro-organism bears such a close resemblance to that of the corn-stalk disease, and the secondary lesions produced very closely resemble those of the contagious pleuro-pneumonia, and as such a disease is very likely to occur in this country it seems well to give Dr. Poels' description in some detail rather than a brief notice of the same.

"In the vicinity of Rotterdam several fat calves belonging to one owner were taken sick at about the same time. The disease often presented a very acute course of from fifteen to fifty hours, and was generally fatal. Some were but slightly ill and recovered. The chief symptoms were loss of appetite, high fever, with accelerated and difficult respiration. Necroscopical examination of the deceased calves gave more or less marked pleuro-pneumonia; *in one case the pneumonia lesion swere so pregnant as to present a characteristic picture of the lungs in contagious pleuro-pneumonia.* The walls of the thorax, as well as the pulmonary pleura, were covered with a fibrinous exudation, as seen in contagious lung plague, but of a much less thickness. The pleura costalis presented many punctiform hemorrhagic centers. The thoracic cavity contained a considerable amount of a clear straw-colored fluid in which floated floculent material as is also seen in the contagious lung plague. In one case this fluid was purulent. The pleura pulmonalis was clouded, and over the consolidated portions thickened and œdematous. In three cases the pulmonary lesions were quite far advanced, especially in the anterior lobe of the right lung. While the interlobular tissue was thickened here and there, and infiltrated with serum, the portion of the lungs mentioned presented a very marked 'marbled' appearance, exactly resembling that seen in contagious pleuro-pneumonia, though the consolidation was of much less consistency. A clear lymph flowed out over the cut service. Mucosa of the larynx and trachea swollen and somewhat reddened; bronchial glands prominent. In the same cases the entire posterior lobe of the right lung was in the same condition as that described for the anterior lobe. *From a patho-anatomical point of view these lungs offered no characteristic which differentiated the lesions presented from those of the contagious pleuro-pneumonia.*

"The external surface of the pericardium was marked by patches of fibrinous exudate, while the inner surface presented numerous hemorragic centers, ecchymotic and diffuse: the sack contained some considerable fluid, the myocardiun had less consistency than normal; the endocardium was marked by numerous ecchymoses. The intestines were but slightly affected, though their walls contained more blood than normal. Mesenteric glands somewhat swollen.

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[*] Fortschritte de Medicin, 1886, p. 388. Baumgarten's Jahresbericht, 1886, p. 158; 1887, p. 124.

"In three other calves which were killed in the acme of the disease the lungs presented nothing pathological, except a few isolated, circumscribed, brown-red centers of greater density than the surrounding tissues. Spleen considerably swollen and doughy. Liver friable. Kidneys and intestines contained an abnormal quantity of blood. Peritoneum clouded. Small amount of fluid in abdominal cavity. The veins of the skin of the dead calves were distended with a thin fluid blood of a dark color. The blood of those purposely killed did not present this appearance, it still coagulated and was not so dark in color.

"The reports of veterinarians and farmers indicate that this disease is one of quite frequent occurrence and the cause of serious economical disturbance.

"Microscopic examination of the organs of these calves, as well as the blood and exudates, revealed the presence in vast numbers of *ovoid bacilli*, which easily colored. With them could be also seen small bacilli resembling those of mouse septicæmia. The ovoid bacilli were easily cultivated, and from the pericardial fluid and the organs and blood numerous pure cultures were obtained. In the thermostat they develop rapidly, while at room temperature they grow very slowly. [Not so with the germs of the corn-stalk disease, which, while they naturally develop very rapidly in the thermostat, still grow fast in room temperature, colonies from blood cultures being apparent to the eye in twenty-four hours. B.] Fatal in calves experimentally. Injections into the lungs caused lesions very strongly resembling those of contagious pleuro-pneumonia in cattle."

Poels confirmed his investigations of 1886 still further the next year, but comes the unnecessary and probably erroneous conclusion that the specific organism of this calf septicæmia is identical with that of the Wild-seuche, Schweine-seuche, etc., though not sure of this.

Here, then, we have recorded evidence that a disease in cattle very strongly resembling contagious pleuro-pneumonia, so far as the pulmonary lesions are concerned, was described in Europe as far back as 1886, and yet we have evidence enough that the British veterinary inspectors did not know of it—in fact, assumed that such a "marbled" condition of the lungs was impossible in any other bovine malady than contagious pleuro-pneumonia. It must be said that such ignorance is inexcusable in these days when the literature of the world is open to any one who seeks it. This is the more inexcusable when we notice that these investigations of Poels' were published in such a universally circulated journal as the *Fortschritte de Medicin*, and then reviewed in that standard work of reference, *Baumgarten's Jahresbericht*, which gives the substance of all investigations, published any where and in any language, each year. These words of censure do not apply to the every-day practitioner, but when a government has a special technical organization, such as the veterinary department of the privy council, it is that department's work to keep its employes posted on all the results of investigation as fast as published. That this disease studied by Poels was not the contagious lung plague is at once evident from its per-acute character, if nothing else; that, in fact, like the corn-stalk disease, it is a septicæmia with lung lesions as a secondary complication was well seen by Poels. The fact that the killed cattle, in an earlier stage of the disease, did not have any marked pulmonary lesions is proof of its original septicæmic character.

It is very singular that so competent and well read an investigator as Nocard should have no knowledge of Poels' investigations, a conclusion we must come to, as he did not mention them in his communication on the American cattle disease last winter nor in a recent communication (*Recueil de Médicine Véterinaire*, Vol. 9, p. 317, 1892), where he reports the investigation of probably the same disease in France, caused by an ovoid organism of the same general character. Nocard, on this account, because of the similar "marbled" condition of the lungs in the French cattle to those seen in the American slaughtered in Paris last winter and the apparent resemblance of the germs found in each case in cultures and their experimental action, at once jumps to the conclusion that the French cattle disease and the American are one and the same thing. He says:

> "Il n'est pas douteux que la broncho-pneumonie infectieuse que j'ai observée l'an dernier a diverses reprises, sur des bœufs de provenance Americaine, existe aussi en France." L. C., p. 321.
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> "There is no doubt that the infectious broncho-pneumonia, which I observed last year in American cattle, exists also in France."

That conclusion of M. Nocard I must positively deny on such evidence as he has given. As has been shown elsewhere, and as seen in Poels' cases, the American disease is not specifically a broncho-pneumonia; it is a Simon-pure septicæmia. The pulmonary lesions are secondary. In all the lungs which have been sent to me since I first studied this disease I have only seen one case (described in this work as the "Lord case") in which there were actual consolidated conditions in the lungs. When the disease becomes chronic the severe pulmonary lesions are a natural result, and the structure of the bovine lung as naturally leads to a "marbled" condition of the consolidated parts.

There is a method of experimentation in such diseases where lung lesions are present as sequelæ that must be absolutely condemned as illogical and misleading. It shows an absolute and astonishing want of pathological common sense. It is the injection directly into the lung-substance of pure cultures of experimental animals. Naturally pneumonic lesions must result. But the real septicæmic nature of the disease is thereby lost. All experimentation should and must be as near to natural methods as possible, to be reliable. Any germ, whether specifically infectious or not, that finds favorable nutrient conditions in the lungs, will cause pneumonia, if directly introduced that way.

As should be well known, I declare, with all the positiveness I am capable of, *that the identity of two apparently similar diseases, occurring in different localities and studied by different observers, cannot and must not be asserted, on the results of the most exact identity in the cultures of the germs in the laboratory, nor by results in inoculated animals alone, especially in the small animals used in such experiments. Poels' results and Nocard's in cattle* also indicate that experimental results in the same species of large animals in different localities and under different conditions, though the germs may be laboratory-identical, must not be assumed as positive evidence of identity with the corn-stalk disease.

No case has ever come up before which so suited me to illustrate my postion by, which is this:

> "*Even though the lesions in the naturally infected species be the same, and the micro-organism causing the diseases be apparently the same, the diseases must occur under exactly the same natural conditions before we dare assert as positively as these bacteriological fanatics do, two diseases occurring in different localities to be one and the same.*"

Before my respected colleague, M. Nocard, who is indeed a most competent observer, is warranted in asserting the disease lately studied by him in France, and which before he did not think existed in cattle there, is identical with the corn-stalk disease, even though he found the same lesions and apparently the same germ, he must show:

First—That corn-fodder (not shelled corn) was in some way connected with the disease in France, for the American disease never occurs in cattle except when fed on corn-fodder, and in this country generally when turned into stalk fields, though, as shown, the leaves on "snap corn" can produce it.

Second—That the French disease is one of a certain season of the year, October to March, the only time when the corn-stalk disease occurs in American cattle.

I dogmatically assert that these two clinical facts are of more importance in establishing or contradicting the identity between the disease studied by Nocard than the pathological lesions seen in both cases in the natural disease, or in inoculated cattle, especially when the cultures are injected directly into the lungs, or the apparent identicity of the germs.

It is very probable that the disease studied by Poels in Holland is identical with that studied by Nocard in France, but it is equally improbable that either are identical with our corn-stalk disease, because they do not occur under the same natural conditions.

At the end of this work is described a new disease under the name of "Summer Septicæmia in Stock," which I doubt not has the same pulmonary complications in protracted cases as occur in the corn-stalk disease and these European diseases, and any but the most careful study of the germs would lead one to assume they were alike in that respect also, though different—the differences being quite marked. But were the germs apparently alike, the time of the year in which the corn-stalk disease only occurs is even a more reliable and positive means of differentiation than any other. Before we are warranted in asserting diseases to be absolutely identical we must be sure that every fact in their history, the conditions under which they occur, their lesions, primary and secondary, as well as their germs, are equally alike.

All Nocard's and Poels' investigations show is that probably one and perhaps two bovine diseases are caused by a member of the same group of germs, botanically, as the corn-stalk disease, to which belong also those of the southern cattle plague in this country; the yellow fever, the ferret pest, and other diseases. Swine plague and typhus abdominalis belong to another group, and the filth disease germ of the German Schweine-seuche, (the government's second germ) and that of Wild-seuche to still another. To the same classes belong hundreds of germs that are not specifically the cause of natural infectious diseases, and still others which are laboratory-pathogenic in small animals, but which cause a natural infection in none.

CHRONIC BRONCHO-INTERSTITIAL-PNEUMONIA IN CATTLE AS A SEQUEL IN THE CORN-STALK DISEASE. (SEE PLATE XI.)

On the afternoon of April 1, 1892, Dr. Lord, a veterinarian practicing in Lincoln, brought to the laboratory a piece of lung from a steer that had died about 5 o'clock the previous afternoon. Dr. Lord was unable to give me much of a history, save that the cattle had been in stalk fields more or less all winter and that three head, this being the third, had lately died after being ill some time and presenting considerable emaciation. It may be well to state that Dr. Lord was very much astonished and alarmed at the condition he found the lungs and strongly suspected the case to be one of contagious pleuro-pneumonia, and that the other cattle must have had the same disease, hence his bringing the lung tissue to the laboratory. A most casual observation of a section of the lung brought in demonstrated that the conditions were entirely different from those seen in lung plague in cattle. In the first place, in this piece of lung at least, there was no indication of a line of capsular differentiation of the diseased portion from the surrounding congested though otherwise normal tissue (as always marks the limits of the disease in pleuro-pneumonia lungs), the same being at the time entirely collapsed retracted, while this piece projected directly above it as a solid wall of tissue. It was somewhat foul and discolored in places and represented a part of the inferior portion of one of the posterior lobes; the pleura covering this piece was swollen and clouded and Dr. Lord reported that the membrane was normal in other parts of the lung and that there was no adhesion present.

The most startling lesion, and the one causing a suspicion of the presence of contagious pleuro-pneumonia, was a more or less general sclerosis of the interstitial tissue of the complicated portions, especially that of the septa separating two minor lobes or sections of the lung, which extended as an uneven whitish band of solid tissue from one-half to one-fourth of an inch in width along the length of the piece; this thickening also extended between the lobuli, but in a more or less

prevailed; and nothing further has been done to settle the matter, though astonishment may be expressed that experimental proof, so easily obtained, was not resorted to."

Strange to say, corroborative evidence in favor of Williams' contention has sprung up in a very unexpected quarter, and on the statement of one of the foremost veterinary pathologists in Europe, who probably knew nothing of what was taking place in England in reference to this matter.

(Were it not for Nocard's investigations we should probably still be told of a new and alarming "contagious disease" in American cattle, had it not been for our investigations in Nebraska, which it seems the Britishers have never read, though their colleges and the veterinary department of their privy council have been favored with complimentary copies. Our Agricultural Department at Washington had also to be taught by a foreigner that there was after all such a thing as the "corn-stalk disease," the "dry murrain" of their own brains having prevented them from coming to a realization of that fact before, though our report has now been published three years.)

The illustrious editor of the London *Veterinary Journal*, after giving the results of Nocard's investigations, resumes upon them as follows:

"Nocard arrives at the conclusion that the disease does not resemble any known in France, and he considers it as a malady special to American cattle; that it is not rare is proved by the fact that since the month of November (1890) he has seen it, always presenting the same features, in other lots of cattle from the United States—Indiana and and Illinois. He is inclined to think that it is the affection known in the western states as the 'corn-stalk' disease, but however this may be, it results from his observations and investigations that American cattle are liable to a microbic broncho-pneumonia hitherto unknown in France, and the serious question therefore arose as to whether any danger was to be apprehended from the importation of such animals. From his observations, Nocard expressed the opinion *that there was no urgent danger nor any necessity for special measures.*

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We know better now, though I am still convinced that we have no disease in cattle which gives to the lungs that distinct, sharply outlined, "marbled" appearance which is now, and ever will be, "*characteristic*" of contagious pleuro-pneumonia. *The condition met with in the lungs of cattle resulting on the corn-stalk disease, while often a chronic interstitial disturbance and somewhat resembling the lungs in contagious pleuro-pneumonia, is so markedly different and generally so washy and broken in outline, in comparison, that any competent veterinary pathologist should be able to recognize it at a glance.* That it can be done is shown by the verdict of Williams and Nocard. In

erally been the *contagious pleuro-pneumonia* of cattle, and I think it can with truth be said to-day that it still remains the only example of a specific and general interstitial pneumonia in any species of animal life. Until very lately we did not know that any process in the lungs of cattle could so closely resemble that disease as can this sequelent *broncho-interstitial pneumonia* in the *corn-stalk disease,* for it is not proper or necessary to that disease, which is a specific septicæmia, but a result in cases of longer duration than investigators would generally come across, or, in fact, have called to their attention in this country. It is only since November last, and again very recently, that I have seen a piece of lung in this disease of the kind so closely resembling *contagious pleuro-pneumonia* as to lead to a momentary suspicion of the existence of that plague. I have seen an immense number of such lesions of a circumscribed broncho-pneumonic type, such as our government veterinarian says were condemned as belonging to the "contagious" plague by the English inspectors when he was in that country in 1880, a few illustrations of which have been quoted. Another fact, apt to be misleading, which has already been alluded to, is this, the lung tissue in the immediate vicinity of these chronic broncho-pneumonic centers in cattle lungs must of necessity generally be somewhat irritated and congested, if not pneumonic, and it is very seldom that even a circumscribed condition of this kind can occur in cattle lungs without more or less œdematous swelling of the interstitial tissue in the complicated parts. Many cattle, otherwise perfectly healthy, come to the slaughter bench with such a circumscribed broncho-pneumonic lesion in the lungs, often very small and sometimes but a single one. They are frequently the only indication left that as calves such individuals have had lung worms and otherwise fully recovered. Mr. Plimsoll, of England, deserves the greatest credit for his humane spirit in calling attention to the abuses cattle suffer on the transatlantic steamships, more especially such as are shipped in the "between decks." The conditions of close air and no movement there abounding must of necessity lead to the aggravation and extension of any such local lesions in the lungs of cattle, and such aggravation and extension would add the lesions of acute pneumonia and increase the interstitial complications in lungs having such weak centers; but even with all this there is no reason that such serious errors in diagnosis should have been made and continued for such a long term of years with no scientific investigation on the part of the governments of Britain and the United States. It is true that this govenment continually protested against the condemnation of cattle and the "bad" name given our whole cattle breeding industry; but these protests were not supported by such evidence as must of necessity be conclusive of their value.

Notwithstanding the examinations and conclusions made by our representatives in 1880, and the positive assertions of Prof. Williams, our chief argument has been *"that there was not and never had been any contagious pleuro-pneumonia in the cattle of the western states."* Our skirts were not free from all suspicion, however. For about thirty years we had to admit that we constantly had the lung plague in our cattle in some parts of our seaboard states, and, as our own government reports show, only too extensively. Hence, that kind of argument was next to valueless. The British authorities could equally well ask (especially in the face of the fact that contagious pleuro-pneumonia had escaped the barriers of the east and broken out quite severely in Chicago in 1886–87, and no one knows how many years it had been there to any exactness): *How do you know it is not scattered in your western herds? What guarantee can you give us that it is not there?*

To say it had "never been seen in western cattle" is no answer to such a question, considering the generally chronic nature of the malady and its difficult diagnosis in isolated cases, and the almost total lack of any well organized veterinary police in this country.

To be sure, the immense slaughtering of beeves at our western cattle marts could be quoted in evidence, and we could with tolerable certainty assert that *"no case suspicious of lung plague had ever been seen,"* but that assertion and such experience certainly was not strong enough evidence to remove a doubt which the British government is naturally inclined to strengthen all it can.

Notwithstanding even the results of our Nebraska investigations and the very conclusive testimony of Nocard and Bowhill in support of the same, still our skirts are not altogether clear of a lingering suspicion and cannot be until every trace of contagious pleuro-pneumonia has been removed from this country.

It will take drastic measures to do this, but it can be done. *Law or no law it must be done.* There is law enough somewhere by which

prevailed; and nothing further has been done to settle the matter, though astonishment may be expressed that experimental proof, so easily obtained, was not resorted to."

Strange to say, corroborative evidence in favor of Williams' contention has sprung up in a very unexpected quarter, and on the statement of one of the foremost veterinary pathologists in Europe, who probably knew nothing of what was taking place in England in reference to this matter.

(Were it not for Nocard's investigations we should probably still be told of a new and alarming "contagious disease" in American cattle, had it not been for our investigations in Nebraska, which it seems the Britishers have never read, though their colleges and the veterinary department of their privy council have been favored with complimentary copies. Our Agricultural Department at Washington had also to be taught by a foreigner that there was after all such a thing as the "corn-stalk disease," the "dry murrain" of their own brains having prevented them from coming to a realization of that fact before, though our report has now been published three years.)

The illustrious editor of the London *Veterinary Journal*, after giving the results of Nocard's investigations, resumes upon them as follows:

"Nocard arrives at the conclusion that the disease does not resemble any known in France, and he considers it as a malady special to American cattle; that it is not rare is proved by the fact that since the month of November (1890) he has seen it, always presenting the same features, in other lots of cattle from the United States—Indiana and and Illinois. He is inclined to think that it is the affection known in the western states as the 'corn-stalk' disease, but however this may be, it results from his observations and investigations that American cattle are liable to a microbic broncho-pneumonia hitherto unknown in France, and the serious question therefore arose as to whether any danger was to be apprehended from the importation of such animals. From his observations, Nocard expressed the opinion *that there was no urgent danger nor any necessity for special measures.*

"The question that now arises on this side of the channel is one of some moment, not only from an economical, but also from a pathological point of view. *Many cargoes of American cattle have been condemned because it was found that one or two in each lot were affected with what was supposed to be the specific and only too well known lung plague.* United States veterinarians have strenuously denied the ex-

istence of that malady among the cattle, and Prof. Williams, when the matter was referred to him on two occasions, has concluded that the lung plague was not present, but that the lesions were those of catarrhal broncho-pneumonia, and now Nocard finds what we are almost forced to conclude is the same disorder, examines it and experiments with it as a scientist of his position and responsibility should do, and, *mirabile dictu*, arrives at the same conclusion as Williams, and even designates it by the same name. *It will be an extraordinary revelation if what has been considered as contagious pleuro-pneumonia in American cattle should turn out to be something very different and of little, if any, importance.* The slaughter mania is a terrible barrier to pathological observation, and little as we have done in the way of scientific investigation of animal diseases in this country, it is to be hoped that the example of France in this instance will not be without effect. It is quite possible that the American cattle found in La Villette market would have been condemned here as infected with contagious pleuro-pneumonia, and the usual consequences would inevitably follow. Must we again say that in this instance, as in so many others, "*they manage these affairs better in France*"? *Veterinary Journal*, Vol. XXXIII, October, 1891, p. 260.

WERE THERE OTHER FORMS OF INTERSTITIAL PNEUMONIA IN CATTLE THAN THE CONTAGIOUS KNOWN TO VETERINARIANS PREVIOUS TO THESE LATTER DAY INVESTIGATIONS?

The fact has already been alluded to, in partial excuse for the mistakes of the British veterinary inspectors, that the general opinion instilled into the minds of veterinary students, and hence, becoming part and parcel of their post-graduate knowledge, has been, and today largely is, *that no other form of interstitial pneumonia, giving to the lungs of cattle anything resembling the "marbled" appearance of the contagious lung plague, ever occurs.*

We know better now, though I am still convinced that we have no disease in cattle which gives to the lungs that distinct, sharply outlined, "marbled" appearance which is now, and ever will be, "*characteristic*" of contagious pleuro-pneumonia. *The condition met with in the lungs of cattle resulting on the corn-stalk disease, while often a chronic interstitial disturbance and somewhat resembling the lungs in contagious pleuro-pneumonia, is so markedly different and generally so washy and broken in outline, in comparison, that any competent veterinary pathologist should be able to recognize it at a glance.* That it can be done is shown by the verdict of Williams and Nocard. In

up bouillon, equally with similarly pure cultures from the plate cultures of the corn-stalk germ from the lung of Dr. Lord's animal. One-fourth cubic centimeter of this mixture was injected into the subcutis of the flank of a full-grown rabbit at 10 A. M. of April 19, 1892; dead the next morning. The greatest precautions were used in opening the animal. The hair over the locus-inoculationis on the inside of the right thigh was first cut and then shaved off; the animal was then soaked in 1:500 corrosive sublimate solution until its entire coat was saturated; the skin over the named point was then cut carefully but quickly through with a red hot knife (It is said my methods are faulty in Washington, though I have never bothered with publishing them before), and with sterilized clamp forceps torn away from its attachments to the underlying muscles. No œdema present! some injection of the vessels, however; small pieces of tissue of a hemorrhagic character were at once inoculated into bouillon, on agar, blood serum, and gelatine plates. The cultures resulting in the tube were predominantly the corn-stalk germ. As that other organism (the government's) grows but poorly, and then only in the first transfer from agar on ordinary solid media—agar and gelatine—and never except then unless quite alkaline, it was interesting to see how many transfers it would take ere it would die out altogether. These agar cultures from the place of inoculation were transferred every three days for about two weeks, and then examined microscopically and by plates. No indication of anything but the corn germ was present in the colonies. That was to be expected, as the government organism is very difficult to obtain in this way, it grows so poorly in such media. Then a rabbit was inoculated and it died three days after—the corn-stalk disease organism only being present in its blood.

Without rabbit or other small animal inoculations this government object can never be safely caught. This made it a very convenient object in their endeavors to upset Nebraska investigations. Of necessity, as well as with good sense, I used and still largely use small pigs in my work on swine disease—always when I desire practical and specific results. Unless the medium is markedly alkaline, and agar at that and exposed in thermostat, one will almost always fail to produce this governmental organism in plate cultures.

Gradually we trace out the ways of these bureaucrats, which are as "queer and strange" as those of the heathen Chinee. *This is why the* *rabbit is used so much and plate cultures in all such instances neglected. They would prove too much. Inoculation of rabbits with lung tissue especially, from most of our domestic animals when diseased with other diseases, will often give this germ only. The germ specific to the disease is thus conveniently lost sight of.* Plates, or plate records, from the tissue originally used, or from the point inoculated in the experimental animal, would be undesirable customers. Hence, a new germ, and singularly the same one, in two diseases at least, and discredit thrown on the work of others. This germ can scarcely be discovered by plate cultures alone except with marked alkaline media and unusual care, so do not seek it in that way. Use rabbits or such animals for that (hum)-bug, but do not forget the plates and to use the organs and blood of the original naturally diseased animal, or you will lose the specific germ in many cases and be led to false conclusions.

The next thing was to test the blood and organs of the rabbit. Blood serum (this is only solid medium that the government organism can be indefinitely transferred on and always grow) and glycerine agar tubes, bouillon, and gelatine plates were at once made from the heart's-blood, liver, and spleen (as said, we did not expect the government thing to grow on the gelatine plates, though once in a while a colony will develop when freshly made from blood or organs).

Four tubes of each medium and four plates of ordinary neutral gelatine from each organ were inoculated. In the tubes the government germ developed nicely. On all the plates but one colony developed, and not a single pollution. This isolated colony was the corn-stalk germ. It grew on a plate from the liver. This shows how easily one can deceive himself, or deceive others, by the inoculation of small animal's with pieces of tissue or other material from animals during a natural infectious disease in which some accidental germ malignant in small animals is present. The specific germ remains local, and the adventitious mixed organism, by its more rapid development and the specially specific and favorable nutritive conditions offered by the small animal (a hundred-fold greater than those offered by the naturally infected one), enters the blood and kills it in a few hours of septicæmia. Forced and unnatural introduction of such a mixture into the healthy organs of the originally natural infected species can only lead to equally misleading conclusions. Such experiments, with the neglect of plate cultures from the animal from which the specific pathologenic germ has been taken, have been recorded, and a new germ reported as discovered, the genuine germ either lost sight of, or not sought after, and discredit thrown on the honest and reliable work of others. I cannot warn young men strongly enough against placing any reliance whatever on results of such tests in small animals, and not too much on the same kind of animals a disease occurs in, when pathological organic material is thus used at once without any plate and other controls. Where the disease is a septicæmia and the natural species very susceptible, such organic material, if absolutely fresh, and the animal killed before complications occur, can be safely used at once for subcutaneous inoculation tests in the natural infected species. If adventitious germs are present, they generally remain local and the specific germ desired enters the blood! These admixtures are seldom present, however, in septicæmic diseases, in the heart's blood, and can, in animals, be generally avoided altogether in our cultures by either using very acute cases, or killing animals in the early stages of the disease. This is sound advice and will stand all attacks from Washington or elsewhere.

THE AGRICULTURAL DEPARTMENT AND THE CORN-STALK DISEASE.

It is an old and trite saying that "you can't teach an old dog new tricks." This axiom is especially applicable to a subdivision of the Agricultural Department in Washington, the method of which has been, ever since its present head had any connection with the publication of investigations, to disparage the work of every other investigator in this country in the same line, unless said person was either in said head's employ, or a tool convenient for use. The method of this model public servant has been to adopt the result of some European investigator's work in animal diseases or follow out some suggestion; to adopt such a germ or description, sometimes either finding an organism in this country suitable to fit the case and at others not, and then asserting the work of other Americans wrong, and that of the Bureau of Animal Industry's only to be the pure and correct thing.

First, we had the micrococcus of Pasteur forced upon us to take the place of the "Bacillus suis of Detmers," which was the correct thing, though not positively proven by positive experiment. This micrococcus swindle lasted from 1880 to the summer of 1886; the report of 1885 issued at that time replacing the micrococcus with a false description of the genuine swine plague germ and a false illustration. That description reads:

"They appear as elongated ovals, chiefly in pairs. This may be due to a greater density or staining capacity of the peripheral portion. The darker portion is not localized at the two poles, as in the bacteria of septicæmia in rabbits, but is of uniform width around the entire circumference of the oval." P. 212.

Again I say that the description is false; that no person who ever knew how to demonstrate the real swine plague germ of this country ever saw it in such character after staining; that the micrococcus organism was false; that the Department of Agriculture swindled the country from 1880 until the summer of 1886, and that it now boldly admits it, for does it not tell us the germ of hog cholera was not discovered until "November, 1885." That is not true as to fact, and also not correct in scientific ethics. A discovery is not made until a true and recognizable description has been published to the world, for this reason every standard European scientific journal prints the day each issue is published on its cover. Taking, then, the universal custom by which all investigators in the world guide themselves, the first correct description and illustration of the true germ of swine plague did not appear in the report of 1885 (issued 1886) but in that of 1886 (issued in the spring of 1887). Therefore according to the world's rule, the government did not discover this organism until the spring of 1887, though it has never conclusively demonstrated its discovery by exact experiments.

The next vagary occurred at this time, a new swine plague, a new germ (luckily they did find a germ that fitted their needs better than the false description of the genuine germ given in report of 1885), because Loeffler and Schütz had discovered such an organism in Germany. The American discovery, after six years' endeavor, still fails to materialize as a fact; that is, as the cause of a specific plague in swine, an epizootic disease. This is the verdict of every competent person who has taken pains to review the second plague question.

Then came my Texas cattle fever work, and the demonstration of its germ. This discovery will stand till doomsday cracks. The Agricultural Department investigators almost immediately took up an assertion of Koch's, that zoöspores will be found to be the cause of nearly every disease germs had not already been found in, and asserted a thing of that kind to be the cause of Texas fever, and ticks to be the transmitter. There is some truth in the tick as a factor in causing this disease, but not in the manner asserted by the government. There is none in the zoöspores as the cause of the disease.

The corn-stalk disease being the discovery of an investigator not in control of the department ring, naturally received the same treatment at its hands. The disease could not be denied, but the germ must be, and something done to discover another to throw a cloud over the reputation of the successes resulting in Nebraska, which placed their own non-success in a bad light before the country.

At the "Nineteenth annual meeting of the Kansas State Board of Agriculture, January, 1890," the chief of the subdivision of the Agricultural Department at Washington said:

> "I do not take very much stock in the germ theory of this disease, because to accept it we must throw aside pretty much of our experience." Report, p. 51.

Why not be honest and say, "I do not take any stock in the author of the germ theory of this disease," as I most emphatically say regarding the author of the above sentence. But that would not do! Some kind of a germ had undoubtedly been found in cattle dying in stalk fields in Nebraska, and some other kind of a germ, capable of forcibly killing cattle, had to be found in the lungs of these animals to offset the effect the Nebraska investigator had had on the public mind in the west. The "ring" must be kept together no matter how rotten the material with which its rents were patched up with. Here we have it.

"*Interstitial Pneumonia in Cattle.*

> "During the past three years three lungs came to the laboratory in which only the pleura and interlobular tissue were affected; the parenchyma was normal or only slightly œdematous. The interstitial tissue was represented by firm, whitish bands from one-eighth to one-half inch in diameter. In these bands were cavities varying from the size of a pin's head to a large pea and communicating with one another. [Note the resemblance to the cyst-like cavities in the same location in the Lord lung. With all due respect to the observers at Washington, I must say that I do not believe that such extensive complications can possibly occur in the lung of cattle and still leave the parenchyma "normal or at the most slightly œdematous." B.] Some were empty; some were filled with a mold of material similar to that of the bands

themselves; some contained a semi-thick grayish mass. The exudate forming these honey-combed bands was partly cellular, partly fibrinous in character. The disease of the pleura was the same as that affecting the interlobular tissue.

"Of the history of these cases nothing definite could be obtained. The importance of determining whether there are types of lung-disease closely resembling the lesions of pleuro-pneumonia, as these did, led to bacteriological investigations of these three cases. In all a micrococcus was found (in the first none other in fact) in the interlobular exudate which closely resembled the swine-plague germ. [Which organism has heretofore beeen spoken of as a "bacterium" in all other reports and hence cannot be a micrococcus in one place and bacterium in another. B.] The general distribution of this germ throughout the exudate in different portions of the lungs leads to the inference that it was the cause of the disease. Its pathogenic properties confirm this view. Subcutaneous inoculation destroys rabbits in less than twenty-four hours.

"Injection of bouillon cultures through the chest wall into the lungs of two calves produced a septicæmia fatal in less than twenty-four hours.

"The bacteria cannot be distinguished from those of swine plague, excepting, perhaps, in one particular. In bouillon cultures not more than twenty-four hours old a distinct capsule may be seen around each germ when the culture is examined fresh in a hanging drop. The bacteria on the edge of the drop do not touch one another and the capsules can be distinctly made out. In subsequent cultures they are not to be seen.

"The significance of the disease and the bacteria associated with it, as well as its relation to pleuro-pneumonia, cannot be interpreted with the aid of the meager material and incomplete histories of the cases examined. The lesions suggest pleuro-pneumonia, while the presence of specific bacteria separates this disease from 'pleuro' in which no bacteria have hitherto been found. It is not improbable that the isolated and rare occurrence of this disease constitutes it a disease of septic origin. The localization of the disease points to an infection from the blood and lymph and not from the air. The bacteriological observations, briefly sketched, will enable us in the future to differentiate this lung disease from pleuro-pneumonia, provided the disease is at its height when the animal is killed and the lung perfectly fresh when presented for examination."

The same objection can be made to the above that Frosch has made against the government investigations of its so-called swine plague, lack of scientific exactness and insufficiency in methodic. No mention is made of plate cultures whatever. It is not mentioned whether the calves were inoculated with cultures made from the rabbits or from the cattle-lungs. I do not desire to be finical, but must object to the intra-pulmonary injection of cultures in all such cases, and if the disease is a "disease of septic origin," and its location points to infection from the blood and lymph and not from the air, what indications were there for such injections into the lungs? Why not try the *via naturæ?* Such experimentation is "bad medicine" to my mind. Then there is the usual "perhaps" and other uncertainties about the work which make it very unsatisfactory.

Furthermore, the government observers assert that they could obtain "no history" in the case of their three lungs. They certainly know the season of the year the lungs were received, and that has a special bearing on the corn-stalk disease. They knew from whence they came and could have easily found out how the cattle were fed, had they been in stalks or not? It is well known, however, that the Agricultural Department does not believe in the existence of the corn-stalk disease or that anything correct can come from Nebraska.

The interesting question now suggests itself, Is the Agricultural Department again up to its old tricks of trying to make an accidental germ take the place of a genuine specific one discovered by us in Nebraska?

What an unenviable yet interesting predicament these people are in. The same germ, or one they can find no means of distinguishing from it, causes not only a second "wide-spread epidemic disease among our swine," a second "swine plague," but also is probably the cause of another very serious malady in cattle, and, most wonderful of all, is springing up everywhere, in cats and dogs, and what not. Why not seek it in themselves, and then test it on their own lungs? We might as well have a *bacillus septicus universalis* causing all the diseases of man and beast, bird and fish, as not. It will save lots of expense and trouble in investigation. The government discoveries certainly indicate that its "germ" is that kind of an organism.

That this swine plague germ of the government, or various non-distinguishable species of the same, so far as rabbit inoculation have any value, is a most ubiquitous character may be seen from the government's special report on its "Swine Plague," 1891, where it says:

> "It will be seen from the experiments given that a germ which is not distinguishable from the swine plague germ was found in the

mucus from the upper air passages of 71 per cent of the cattle examined; 85 per cent of the cats and 33 per cent of the dogs from which inoculations were made." P. 155.

Let us see if any "interstitial pneumonia" is mentioned in any of these cattle:

"Rabbits were inoculated with the mucus taken from the larynx of seven healthy cattle. Four of these were steers, two of which were western cattle shipped to Washington for beef. The four steers were killed in a slaughter house near the experiment station. The other three were heifers killed for various purposes in a similar manner at the experiment station. In each case the mucus was collected immediately after the death of the animal and inoculated subcutaneously into rabbits." P. 151.

It will be remembered that the government says, that apparently the same germ, in the "interstitial pneumonia cases," worked "through the blood and lymphatics and not from the air." How did it get into the blood?

There are but three possibilities: First, by way of the digestive tract; second, by way of the respiratory tract; third, by means of the cutaneous wounds. If the germ is in the mucus of the larynx it must get there either by means of the air, or by the mouth; in the one case it can get into the lungs in the same way,.and the other into the intestines, and if some other cause has produced disease in the lungs it can get into the "blood" and "lymph" systems, and if there are lesions in the intestines it can also get into them both. It has been shown that the same germ was in the lungs of a steer undoubtedly diseased with the "corn-stalk disease;" the germ being demonstrated by rabbit inoculations, the corn-stalk germ by plate cultures and control cultures on different media. If they had taken this germ found in the larynx of otherwise healthy cattle and injected it into the lungs of healthy cattle they might have found it would also have produced pneumonia if small enough doses had been given. The question is still open, Does it of itself cause disease in cattle as a specific infection natural?

Now it may be said that Nocard did not find it in the cattle lungs examined by him. He might have had he inoculated rabbits before he made his plate cultures and then might have lost sight of the true germ. He can have easily missed it in his cultures, for from about three years' experience with this germ I know it will not grow in the same media that the swine plague, corn-stalk, and several other such germs will, and that it requires a degree of alkalinity in the medium, to develop which materially interferes with the development of the true swine plague germ and many others. Others have noted this peculiarity of this germ or bacteria which cannot be differentiated from it. I always have used a very delicately up and down neutral medium with no shade of alkaline in it for cultivating my swine plague (hog cholera—government) germ, because experience has taught me that it keeps out the possibility of many other organisms developing and is thus of much practical value.

It is self-evident that the "specific germ" of the Agricultural Department, being all about, does nothing of itself, some other germ or factor must first prepare the way for it. It is a handy complicating occurrence, that is all. An inspector of the government and a man who should be able to speak with authority on its being the specific cause of a special plague (or even disease) in swine, distinct from swine plague proper (the government's cholera), said at the meeting of the United States Veterinary-Medical Association, held at Washington September, 1891:

> "It has been my opportunity for the last three or four years of doing some work in that line myself, in association with Prof. Welch, of Johns Hopkins University. I might say, in parenthesis, that my work in this line has nothing to do with my position as government inspector. As to what connection the organism (Salmon's swine plague germ) has with the lesions described in the reports of the bureau, is a question on which we all might not agree. Nevertheless the swine plague organism does cause trouble. The trouble in hogs is, as a rule, in our experience, one of mixed infection. We have not had the opportunity of seeing an outbreak of swine plague (Salmon's) pure and simple. We have found that it is very hard to say when swine plague (Salmon's) is present that hog cholera (Salmon's) is absent, from the fact that swine plague kills [What, rabbits or hogs? B.] in a few hours, while hog cholera requires some days. If, then, an animal be killed and presents lesions in the intestines, as are generally supposed to be characteristic of hog cholera, the statement must be very carefully considered before it is made, that hog cholera is not present. We were thrown off our track during the earlier part of our investigations. We found afterwards that hog cholera did exist in these animals that we thought had swine plague (Salmon's) pure and simple." *Journal of Comparative Medicine*, Vol. XII, p. 549.

It will be interesting to see the gyroscopic somersaults of the bureaucratic investigators on this question, in the face of all the evidence collected in this contribution to the history of the corn-stalk disease. They will doubtless regret their too hasty publication of the cattle-lung investigations reported above, or their neglect to use all the laboratory methods necessary to exactness. They may even yet learn that Billings has not been so very far wrong when "he boldly announced a great many discoveries that have (fully) fulfilled the expectations of his friends." Answer to Dr. Peters, p. 35, *Journal Comp. Med.*, Vol. XIII, 1892.

THE ETIOLOGICAL NATURE OF THE CORN-FODDER DISEASE.

Like most of the infectious diseases which seriously interfere with the prosperity of the live stock interests of the United States, this "corn-fodder" disease has nothing of a contagious character about it. It does not owe its primary origin to the presence of a diseased animal in the first place, or of any material from such a diseased animal among healthy stock; it is not an endogenous (generating from within) or obligatory parasitic disease. On the contrary, a better example of a facultatative parasitic or exogenous (owing its genesis or origin to external circumstances) disease could not possibly be found. It most aptly illustrates my continuous endeavors to impress upon the medical world the absurd folly of the continued use of the words contagious and infectious to express class differentiation in disease. The word "contagious," used according to its roots, simply expresses to us the fact that the infection of a healthy individual in a given disease either took place or can take place by means of contact either with an individual diseased with a specific disease or with some material directly from such an individual. Historico-pathologically speaking, the word contagious only means that the primary origin of the inficiens (infecting material) was within the body of an already diseased animal. Any other definition of this word is absurd; but the terms which I have selected to express this class of diseases, while equally as technical as that of Pettenkofer, "endogenosis," is more self-evident to the ordinary class of the readers, viz., "intra-organismal"; that is, originating within or from a diseased animal organism. On the contrary, I have called the opposite class "extra-organismal diseases," simply because their primary origin must be invariably sought in the external surroundings or conditions. In the disease in question we have clearly demonstrated that its primary cause is to be sought in the corn-fodder, which certain susceptible animals eat.

Again, this disease bears a very close relation to both the southern cattle plague and the swine plague, in that it is an absolutely local disease. All practical experiences of farmers demonstrate the fact in *optima forma,* for they all tell us that it is the corn-fodder in a certain field which is dangerous to cattle, and not all corn-fodder by any means. It is the same thing with regard to the disease in the corn itself, and I feel sure that more exact study will eventually demonstrate that in many cases this localization, with reference to disease in corn, will be found so centralized that even a single stalk in a single hill, or a group of stalks, will alone be found diseased, while in other cases there will be several, and in still other places whole groups of stalks, or even portions of a field or the major part of it will be complicated, as witnessed in the "forty acre" field previously alluded to.

THE PATHOLOGICAL NATURE OF THE CORN-STALK DISEASE.

In diseases of this character it makes a tremendous difference at what stage of the disease one makes an examination of a sick animal as to what conclusion he would come as to its nature, judged by the pathological lesions which would be presented to him. To those who have read this article with any degree of care, especially the parts referring to the lesions observed in American cattle in England, and Nocard's observations, may, with the British inspectors, Professors Williams and Nocard, have come, to the conclusion that they did, that the disease is a "broncho-interstitial pneumonia." That determination would be justified when what is before the eye alone (and such phenomenon too often rule the judgment, and as often lead to erroneous conclusions) is taken into consideration; or better, the most striking phenomena, but not all by any means. It is doubtful, perhaps, if in these cases the whole pathological picture has been presented to the European observers. It must be borne in mind that up to this year that I had not seen any such conditions in the lungs as they had seen in Europe; in fact, I have not seen a case in the fields, and it is due to a sort of fortunate accident that I have been enabled to confirm their observations, so far as the lesions in the lungs are concerned this year, through the interest and kindness of Dr. Lord, and the piece of lung sent from Humboldt, Nebraska, in alcohol. The case killed and examined by Dr. Thomas among Mr. Delaney's cattle in 1889, however, gave evidence enough that not only pneumonia, but pleuro-pneumonia, occurred as a complication in this disease, though bearing no resemblance to contagious pleuro-pneumonia, so that in the first publication on this disease I wrote on this question:*

> "Were I to be asked what disease of animal life this corn-fodder disease most closely resembles, I should say it is the exact counterpart of the genuine swine plague, and, in fact, most respectfully refer the reader to page 320 of that report. In the corn-fodder disease in cattle, every organic lesion and every variety of lesion seen in swine plague will be seen, with the exception of the ulcerative and neoplastic lesions, and perhaps diphtheritic, seen in the intestines, the two former of which are primarily due to idiosyncrasies of structure in the hog, and only secondarily to the action of the bacteria causing that disease. Here, too, we have the same excessive parenchymatous changes in the great glandular organs of the body common to all acute diseases of this character. Here, too, we have pneumonia in all the various types seen in swine plague, and again, one form bearing the very closest resemblance to those in swine plague, marked by the engorgement of the interlobular and bronchial vessels and coagulation of the blood within them, which I illustrated in Plate XIII of my swine plague report (Plates I and II of this), and which is equally well applicable to this corn-fodder disease. In both species of animals the pregnancy of this lesion is due to the peculiar loose and open character of the interlobular tissue and the large vessels circulating therein. Again, in both diseases we may find an acute broncho-pneumonia, not due to the entrance of the specific germ *via* the respiratory tract, but to the extreme degree of interference which the circulation suffers, and the consequent effusion of serum into the air tubes, especially the smaller, obstructing them and leading to atelectasis and pneumonia. I neglected to call attention to this variety of consolidation in my report on swine plague, but it is really a very common form, and to be easily distinguished from that variety caused by the entrance of the germs *via* the respiratory tract which is more acute, more multiple, more rapidly caseous, and especially not hemorrhagic in any of its parts as in the surrounding tissue, and also begins immediately around the tube, whereas this form begins in the more distant part of the bronchiolic territory."

The first report on this disease was sent to all the veterinary colleges in Britain and Europe, in which, as can be seen, the existence of

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*I have not yet seen the sclerosis of the interlobular tissue in swine, now known to exist in this disease, but that may be because I have never examined any very chronic cases of swine plague.

"pneumonia" was most distinctly stated, and in which Dr. Thomas says: "Pleuro-pneumonia sufficient to kill; the entire lungs congested and the lower portion of the lobes solid." (P. 184). Yet in his vain endeavor to uphold his crumbling reputation as a pathologist, and futile efforts to upset Prof. Williams, we find Walley quoting a two-page article by Dr. Spencer on this disease, being a short paper read before the California Veterinary Association. It is evident from Spencer's paper that he made no pretension of knowing anything of the disease personally and simply made a digest of our investigations in order to introduce the subject to his colleagues there present. No thoroughly honorable physician would quote such authority as that, when the original was at command, in order to make a point in discussion against a professional opponent.

Walley says: "The theory that the case of the Deptford bullock was one of 'corn-stalk disease' is anything but a happy one, in the face of the description of the symptoms and post-mortem lesions given by writers on the subject. Thus, in the *Journal of Comparative Medicine and Veterinary Archives*, February, 1892, Mr. H. F. Spencer gives an account of the malady, which shows that it is of the nature of a septic-intoxication, and mentions that congestion of the lungs was found as one of the pathological phenomena." (McFadyean's Journal, March, 1892, p. 55.) I have sections of that "Deptford bullock's" lung, stained by Dr. Bowhill, in my possession at this moment, as well as his photographs of the same section, and in both can be seen the ovoid-belted germs, and the lesions are essentially the same as those described here in the lung from Humboldt, Nebraska. In no natural case of the disease, the organs of which have been sent to me, were the lungs absolutely free from complication, and are so described; but consolidation of lungs may not be considered "pneumonia" in the "Dick Veterinary College," though it has been designated "contagious pleuro-pneumonia" in American cattle often enough by the same authorities.

Let me say here, that I cannot possibly look upon all the cases of pneumonia in our cattle that have been killed and condemned in Britain on account of contagious pleuro-pneumonia as belong to the "corn-stalk disease" by any means. I do not think one of the cases, as described in the report of our Agricultural Department, 1880–81, to which reference has been made, as belonging to this disease. As I have said, they represent chronic, circumscribed broncho-pneumonia, which has been superseded by acute complications contracted on board ship. I must also think it is about the same with those cattle examined by Nocard. That is, that they had a mild attack of the cornstalk disease in this country and would have, in all probability, recovered had they remained here, but that they were shipped at just the right time for the shipping influences to aggravate the trouble and complicate it with an acute and fatal form built upon and around the previously diseased pneumonic centers acquired in this country. We do not know all about the lesions possible in this disease yet, but with the aid of the investigations of Nocard and Bowhill, supplemented by my own of this year, those who have any pathological acumen should certainly be able to build up the entire structure and comprehend all the possibilities. The corn-stalk disease is very much like *typhus abdominalis* in man. That disease is a septicæmia, the lesions in the small intestines (Peyer's plagues) being like those of swine plague due to structural, that is, a racial-anatomico-physiological idiosynocrasy in swine or man respectively. As in *typhus abdominalis*, a "typhoid pneumonia" is only too often a frequent and unwished-for sequel, so we have in this corn-infection-septicæmia a pneumonia, varying in character, as a sequel.

THE DISEASE ITSELF IS A SEPTICÆMIA.

Prof. Williams seems to have a far clearer idea as to how a pneumonia can follow, as a complication, in a septicæmic disease than his energetic business rival. (Business knows no courtesy. Professional ethics once taught that courtesy should be a distinct characteristic of the physician. The faculty of medical colleges, run on business principles, should not always be considered as responsible, however. Professional ethics are strangers to such institutions in this country and probably in Britain also. The ungentlemanly and discourteous principles of the faculties have permeated the majority of the alumni, and the practice of medicine in this country has become as "cut throat" a business as is the practice of catching students in the colleges. How is it in Britain?)

On looking over the literature of "American pleuro-pneumonia" in Britain I came across the following from Prof. Williams, on the initial lesions of "broncho-pneumonia," which I introduce here, as I desire to draw attention to the initial lesion in the lungs in the corn-stalk disease and its effects on the peculiar character of the lesions in those organs in some cases while not in others. Williams studied these lesions in connection with Prof. Hamilton, and it is the latter whom he himself quotes. It is interesting to note that the studies were also made on the lungs of American cattle:

> "On careful comparison, however, of many cases we feel assured that the first deviation visible is a relaxation and distension of the abundant plexus of blood-vessels ramifying in the inner fibrous coat, immediately beneath the basement membrane; that is to say, of the branches of the bronchial artery. They became engorged with blood, so that on transverse sections they appear like little cavities distended with blood corpuscles. In a few hours afterwards the basement membrane becomes more apparent than it usually is, and at the same time more clear and homogeneous, while the surface is thrown into many folds. These changes in the basement membrane are apparently due to its becoming œdematous, serous fluid being infiltrated into it from the underlying plexus of distended vessels, and we shall see that, as the acute irritation continues, this œdematous state of the basement membrane becomes more and more a well marked feature. The next change, so far as we have been able to calculate, occurs in from twenty to thirty hours after the primary distension of the vessels and consists in the loosening and desquamation of the columnar epithelium at the foci of greatest congestion. The columnar epithelium is thus shed at a very early stage of the attack and takes no part whatever in the after changes which ensue. The cause of this desquamation of the columnar epithelium seems to be the œdema of the basement membrane loosening its underlying attachments very much in the same way as the vesicles, which form in an acute inflammation of the skin, loosen the attachments of the superficial layer of epidermis. And, furthermore, it can be easily understood that where the desquamation takes place in an inordinately great extent the loss of the ciliary action of the columnar cells will seriously interfere with expectoration and tend to cause the catarrhal products to gravitate downwards towards the smaller bronchi and air vesicles." Department of Agriculture, "Contagious Diseases of Domestic Animals," 1880–81, p. 236.

The above remarks will serve very nicely as an introduction to my own on the very same subject, and hence I need not repeat them, but will discuss points bearing more directly on this disease. Although it is probable that we have not the entire pathological picture of the corn-stalk disease and its possible complications as yet presented to our view, still we have seen enough to enable us to build up a tolerably complete structure and perhaps anticipate any further complications that may arise. Attention has been previously called to the fact that both Professors Williams and Nocard looked upon the disease as a "broncho-pneumonia," the former designating it as a "catarrhal broncho-pneumonia." I have also said that I could not consider all the cases of broncho-pneumonia of a chronic circumscribed type, to which an acute ship pneumonia has supervened, to belong to the corn-stalk disease, though perhaps every case in which extensive interstitial processes have been added may be cases of that disease acquired in the winter months here, and which, had they not been exposed to the vicissitudes of transatlantic shipment, would have recovered had they remained on their native heaths. Whether or not the disease is non-recurrent I cannot as yet say. Again, whether or not in recovered cases the local pneumonic centers in the lungs become encysted, as in contagious pleuro-pnemonia, cannot be answered at present, but it seems to me that such is highly probable. Such questions as these can only be settled by very careful observations and bacteriological investigations and experiments at abattoirs, for in laboratories like this we never come in contact with chronic (recovered) cases in such diseases, only the acute and fatal ones coming to our attention. The mistakes in diagnosis in Britain, the successful results following Nocard's and Bowhill's investigations, and the almost positive certainty that all the cases condemned in England as "contagious pleuro-pneumonia" could not possiby have been even sequelæ of the corn-stalk disease, almost emphatically point to the absolute necessity of the establishment of small but completely equipped laboratories, with a suitable building for all kinds of animals, and a competent corps of investigators, not only by our government at the great slaughtering centers, but by Britain and every country that has not such institutions. Had we had such institutions at Chicago, Omaha, Kansas City, St. Louis, and perhaps Boston, this unfortunate muddle would have long since been closed up. They are absolutely essential to the welfare of our great live stock interests and the best development of our export trade in stock cattle on the hoof. We must absolutely know every possibility of infectious diseases in our stock so that we can give the world true and exact accounts of the same that they may be judged exactly as they are. The evidence must be exact and conclusive, and of such reliability as never to be open to question.

To return to our corn-stalk disease:

We will consider it principally according to the evidence as furnished by our own investigations:

First—We have the case of the inoculated calf, January 27, 1892. This case truly represents the disease as it is, in its simple uncomplicated form. A septicæmia, as it occurs in the majority of cases as reported by farmers, where the cattle scarcely show any signs of illness and die in twenty-four to forty-eight hours after being turned into stalk fields.

Second—We have the Fremont cow case, reported by Dr. Thomas and published in the first edition of this work. This case represents a more prolonged condition of slower infection, in which great circulation disturbances have begun and the pneumonic complication is in its early stage of development.

Third—The "Lord case," which represents a still more advanced stage and slower course, with full development of the pneumonic complication, showing one essential character of this sequel, that is, the necrotic processes in the lung tissue.

Fourth—The "Humboldt case," which represents a still more chronic development of the pulmonary lesions, with no tendency to necrosis and less severe circulation disturbances and complication of the parenchyma, but more even sclerosis of the interstitial (interlobular) tissue. In the last respect this case bears a closer resemblance to contagious pleuro-pneumonia than either of the others.

The whole picture is very easy of comprehension when one constantly bears in mind that *sui generis*, the corn-stalk disease is a septicæmia, and compares it with diseases of the same character and the pulmonary complications occurring in each, viz., *typhus abdominalis* and swine plague, both of which are complicated by pneumonia and yet pulmonary complications are not essential to either. In swine plague it is more common than either in the corn-stalk disease, or in *typhus abdominalis*, first, because the physiological conditions of the circulation in swine are far less favorable than in cattle or man; second, because swine are far more exposed to the action of adventitious germs in the lungs, which come into play after the swine plague infection has prepared the field; and third, because, in swine, direct infection by aspiration of the specific germs through dust is not at all an uncommon occurrence under favorable conditions.

The corn-stalk disease is a specific septicæmia, the infection invariably occurring, under natural conditions, by way of the digestive tract. In all such diseases we have, in all probablity, three essential factors to deal with, in some far more than in others. These factors are:

First—Fever, due to the action on the choleric centers, of some product of the germ.

Second—The effects of fever on the tissues.

Third—The effects of some toxic product of the germ on the parenchymatous tissues, thereby augmenting or supporting the action of the fever on the same elements.

The influence exerted by the two essential factors on the parenchymatous tissues is not always equal in all diseases of this kind. In some the fever alone exerts the chief influence and then the parenchymatous changes have not the tendency to necrosis that they have when the toxic action on them is of equal severity. The great influence, however, exerted by both these factors, is on the circulation. To these alone are principally due the pulmonary sequelæ so frequently observed, and the character of the latter is dependent on the severity of the former. The pulmonary lesions in the corn-stalk disease are a most striking example of this fact.

As a result of the fever and toxic action on the dense parenchymatous organs which includes the muscles of the body, the liver, the kidneys, and above all, the heart, we have an intense swollen condition of the cellular elements of those organs, extending to fatty degeneration and even necrosis in many cells, which produces an anæmic condition in the same. This condition is again favored by the proneness of the diseased individual to inaction and the consequent non-stimulation to the circulation. The parenchymatous cells have become their own enemies. They may want food, but they cannot get it if they would. The weakened heart can no longer force it to them, and their own swollen condition has so pressed on the delicate nutritive-canals as to form a dam, so that the blood cannot be forced to them, even though the heart had the ability to do it. What is the natural result of all this disturbance to the circulation?

A stocking of the blood in those parts the anatomical structure of which is such as to offer the least support to the walls of the blood vessels; that is, those parts allowing the greatest distension of the blood tubes. Which are those parts or organs?

First—The lungs in the first place, but particularly those parts anatomically so situated as to favor such engorgement of the blood vessels. The dependent portions in the first instances, and those more distant from the heart, the anterior lobes, in the second.

Second—The vessels of the mesentery and the walls of the intestines.

The circulation disturbances in the lungs alone interest us at present. To the mere reflow, or stocking disturbances, in the vessels in the sections of the lungs named we have also to remember the continued influence of a weak heart. Whatever else may be connected with the process of coagulation of the blood in the vascular system, we do know that one great essential to non-coagulation of this vital fluid is a normal condition of the intima, or lining of the blood vessels, and that a continuous and rapid circulation of the stream is a necessity to this condition of the membrane, for its elements derive their nourishment from the circulating blood. The moment the circulation begins to stagnate these cells suffer, and complete stagnation means their death, and the intima becomes a foreign body which at once favors the coagulation of the blood in several vessels. But we have another factor to deal with as well, and in this case it is the specific bacteria which are multiplying and accumulating in the stagnating stream, and offer so many billion foreign bodies around which the blood begins to coagulate, and to this must be added, in this disease at any rate, the effect of the products of these germs on parenchymatous cells, though this seems to vary in different cases. The lungs of the calf inoculated January 29, 1892, very perfectly illustrate these conditions before complete coagulation in any part of the circulation in the complicated portion of the lungs had taken place. (Plates I and II.) There is a general distension and engorgement of the blood vessels. Such a general disturbance is almost immediately followed by some disturbance of the intima and exudation of blood serum and leucocytic migration from the vessels into any cavities present and on to any surface, as into the alveoli and bronchial tubes of the complicated parts of the lungs. This is the condition described by Prof. Hamilton (quoted by Williams) as that of the initial stage in capillary bronchitis, and I need not therefore refer to it again. This is not pneumonia, not even catarrhal pneumonia, though it may soon lead to that, for the moment this catarrhal condition and these disturbances of the circulation have continued long enough to cause acting proliferation in the cells lining the alveoli, and the neoplastic cells are exfoliated and added to the serous-leucocytic effusion, then we have inflammation—then catarrhal-pneumonia begins.

As demonstrated in the first edition of this work, this form of pneumonia, under these conditions, does not begin in and around the bronchioles, but in those lobuli most distantly situated from the bronchioles, the cellular accumulation, consolidation, extending from without inwards; that is, from the peripheries of a bronchiolic territory toward the tube itself. The reason for this is simple and easy to be understood. The so-called walls of the alveoli are but the extension, or continuation of the adventitia of the bronchioles plus the reticular circulation system and a very microscopic amount or remnant of the muscularis; with a single layer of pavement epithelium, which in reality forms the walls of the alveoli, and not the so-called "septa" proper, which are but supports. The ciliated epithelium of the bronchioles has been changed, by adaptation, to the single-layered pavement epithelium of the alveoli. The mucosa stops where the bronchiole ends. The mucosa of the bronchiole is supplied by an even more delicate network of blood vessels than surrounds each alveolus, and on disturbance of the circulation as above described, the same conditions prevail; distension and engorgement of the blood vessels and exudation into the tissues of the walls of the air-tubes, swelling them up and into the lumen of the delicate bronchioles, stopping them up so no air can pass in or out. The supply of air thus shut off more or less rapidly, with the accumulation of the exudate into the tube, must be felt first and most severely at the most distant parts of the bronchiolic territory; hence, here the alveoli are the least extended with air, and here occurs the first want of support of the blood-vessels; here is not only the first serous-leucocytic effusion into the air cells, but also the first circulation disturbances begin, and hence here, also in these peripheral alveoli, the pneumonia, in these cases, the filling of the alveoli with leucocytes and neoplastic-exfoliated cells, first begins, and extends or develops central-ways towards the bronchiolus itself.

I do not think this initial lesion in the development of pneumonia of this type (origin) has been sufficiently well understood, probably because most of the investigations in pathology have been made by

M. D. pathologists on human beings, and the latter seldom, if ever, offer opportunity to study initial lesions. This is not the broncho-pneumonia described in the books, and yet it is also a "Herdweise" (center-developing) broncho-pneumonia. That other form, the "orthodox" variety, originates exactly in a contrary direction to the above. It begins in the bronchial tube and extends from it. In fact it also must, almost immediately, begin peripherally as well, and the two processes extend towards each other until they meet. In the initial stage only are they different.

Let us go back to our capillary catarrhal bronchitis; an effusion of serum, plus leucocytes, into the lumen of the bronchioles. Before we go further, however, let us do a little nature worshiping and give our attention to one of the most wonderful adaptations of elements, to use, that nature offers us. Let us consider, briefly, the comparative evolution of the respiratory tract, for unless we do so we cannot comprehend the wonderful machinery of mammalian respiration. I remember once, when examiner in a certain medical college in this country, asking a would-be graduate, "Please tell me all you know about pneumonia?" The answer was, "Inflammation of the lungs, sir." The next question was, "What, then, is inflammation of the lungs, young man?" Answer: "Pneumonia, sir!" That was really all that student knew about it, except, had I questioned further, he might have repeated such hackneyed expressions as "red-hepatization," "gray-hepatization," or even "yellow-hepatization," "resolution of the pneumonia," and such stereotyped technical terms which the majority of medical graduates in this country fully as well comprehend as did that student understand what pneumonia really was.

The respiratory apparatus is an evolution out of the alimentary, a development out of the œsophagus at its anterior termination. It is formed of the same coats. In the beginning a node-like projection extends from the anterior wall of the digestive tube and soon enlarges, then attains a bifurcature in its free end, and so on develops, indications of bifurcation appearing continually on its outside surface. At first this node is solid, but even then it will be found composed, from inward-outwards, of an epithelium, a muscularis, and a serosa, and soon something else, a tissue that is not in the œsophagus, appears, a phylogenetic-atavistic remnant, eminently useful, the tissue to make an open, non-collapsing tube, which is not in the œsophagus, the reminder of the days when our ancestors crawled on their bellies, and swam in the seas or rivers—when we had gills, mere bronchioles—the cartilages. But it is with the mucosa and its ciliated epithelium only that we have to do. The epithelium of the œsophagus is pavement and its mucosa supplies the natural lubricant for the bolus to glide over; the food must go down easily. The lungs want none of that. "Foreign-body-pneumonia" would be the common result; in fact no one would live were that same structure continued from the matricial œsophagus into the secondary respiratory tract. Here nature had to provide for protection. Here all foreign enemies must be caught and driven out. Here must be stationed a guard, more subtly powerful, greater in number, than the combined populations of the world. In nature's omnipotence, one life is more needful of protection than all the combined wealth of the world. To meet this necessity by a process of evolutional adaptation (I positively deny all purpose in nature), the parent pavement epithelium of the œsophagus becomes changed to the "armed" ciliary epithelium of the air tubes, which, like billions on billions of microscopic bayonets, stand with ever presented arms, from the microscopic termination of the bronchioles to the larynx, waving, waving night and day in one direction, from the "door to life" the air cells, to the catch-basin for the expectoration, the pharynx, wafting back each microscopic foreign body in the aspired air that otherwise would carry with it death and destruction to the individual. But this is not all; these particles in the aspired air must be caught in order to be moved by the protecting cilia; hence the mucosa which covers these delicate hair-like terminations of the epithelial cells (the evolutional beginning of the terminal nervous system) with just viscidity enough to catch all foreign particles, so that from cilia to cilia they are passed on and upwards where they can no longer be dangerous, and finally expectorated. But more, not all dangerous elements come from without. Any irritation of the delicate net of capillaries nourishing the mucosa causes an extra excretion (secretion) of its products; the very element by which protection is insured can itself cause disease, but so long as the product is not beyond the means of the cilia to waft on and out life is safe and disease prevented. But the product can be too great, and this is just what occurs in the conditions we are considering at present in the lungs of cattle in the early stages of the corn-stalk disease. In this stage of catarrhal bronchiolitis, the effusion is so watery and so plentiful that the cilia become "drowned out" like a "drowned rat," if I may use the expression. They can move no longer. Now the most common of all foreign particles in the aspired air are bacteria. Under normal conditions in the respiratory tract, they are harmless. They are caught in this natural fly trap and wafted out again. But with the cilia drowned out or down, and with blood serum at that, and the favorable temperature of the body, we have the most favorable conditions possible to the development of many germs (not all, thankfully), and if such germs are thus caught in this blood serum in the bronchioles, they at once begin to develop and in most cases set up a mechanical irritation, to which in some is added a chemical irritant, and in very few a toxic influence. In one way or another, or all combined, the irritation extends to the mucosa, then the submucosa, and finally beyond the walls of the bronchioles to the parenchymatous tissues of the lungs, and leads to the paradigmatic, orthodox broncho-pneumonia, which will take a mild or malignant character according to the toxic, or not, character of the germs which were its active cause. Thus in the corn-stalk disease, we first have the broncho-pneumonia due simply to the serous obstruction and shutting off the air in the bronchioles, beginning in the peripheries of the tubes, and to this is superadded the broncho-pneumonic centers beginning within the tubes themselves and extending from them. It depends whether or not micro-organisms are at once caught in the tubes which antedates the other and predominates the character of the disturbance. The variety beginning in the peripheries of the bronchiolic territories is clearly and almost uncomplicatedly illustrated in the sections of the lungs of the inoculated calf, while both forms are as closely illustrated in similar sections from the lungs of the Fremont case. (Plates I, II, and XI.)

Any one who has the opportunity, and who carefully studies the microscopic lesions in the lungs in the different stages of the corn-stalk disease, will surely convince himself of one fact, which is, that the initial lesion in the lungs is an engorgement of the blood-vessels and stagnation of the circulation in those parts, anatomically so situated as to be favorable to such pathological conditions. As already indicated, the complication of the heart-muscle and its consequent weakness aids and abets the above conditions. According to the severity of the general parenchymatous disturbances over the body is the rapidity and severity of these circulation disturbances in those portions of the lungs favorable to their development; and dependent on the same phenomenal conditions is the rapidity and completeness with which coagulation occurs in the distended vessels and shutting off of more or less tissue from the circulation, and the tendency to and completeness of the necrotic or necrobiotic conditions in the diseased parts.

When congestion takes place rapidly and completely, and this occurs at first in the most dependent centers, or those where the natural conditions are the least favorable to the circulation, and in this disease the bronchial arteries seem to be frequently the first thus obstructed, we find scarcely any tendency to neoplastic conditions; on the contrary we find an almost immediate necrosis, a sort of pathological melting down, of tissues, and the formation of circumscribed necrotic centers marked by the presence of patches or masses of a hyaline substance staining diffuse blue in properly treated hæmatoxyline sections; these centers are immediately connected with and surround the bronchial tubes; the walls of the bronchioles, and frequently their contents, as well as the septa of the alveoli in their immediate vicinity, also undergo this hyaline degeneration. The centers in the "Lord case" most perfectly illustrate this condition.

As noticed in the description of sections of the lung from that case, there is no evidence of neoplastic processes in these centers, whereas, on the contrary, they are very marked in the walls of the alveoli and around the vessels in the tissues around these necrotic centers. This fact seems evidence enough that the most severe and acute disturbances of the circulation in such cases must be in the bronchial arteries, and this is supported by the fact that the circulation in this system is much more unfavorably situated for rapidity and heart-support than the balance of the pulmonary circulation, which is far more directly opposed to the immediate action of the propelling energies of the heart. The entire circulation is, in the diseased portions of the lungs, however, eventually more or less severely complicated. But the necrosis is not limited to bronchial arterial circulation by any means. Later on it extends to the more distant tissues. Attention has been previously called to the fact that the primary disturbances in the parenchyma frequently begin in those alveoli most distant from the bronchioles and extend towards those tubes; in other words, that the consolidation begins there first, and hence is more intense there earlier than in the central part of the bronchial territory.

It will not be disputed that the more intense the consolidation, the more the distension of the air-cells with proliferated cellular material, the more anæmic does that tissue become, the poorer its nutrition, and the greater the tendency to necrobiosis and destruction of the elements, not only the cellular infiltration, but the septa of the alveoli; this fact is again proven in the Lord case, where the entire mass filling the alveoli is dead and more or less loosely attached to the walls of the alveoli, the necrotic mass could often be very readily removed *in toto* as dead tissue, and in some cases portions of the septa came away with it. In others the greater number of the cells in the center of the mass filling the alveoli were in a condition of "coagulation necrosis" amidst much granular detritus. This same disturbance of the circulation, with the final result of coagulation of the blood in the vessel, often leads to complete occlusion of the smaller blood vessels, leading to inflammatory processes in their walls and around them, which transforms them into cords of sclerotic tissue or striæ of the same, much wider than the original dimensions of the vessels; this often leads either to necrosis of the lung tissue in such spots or more frequently to the formation of quite extensive centers of sclerotic tissue. The one or the other depends entirely on the rapidity with which the disturbances of the circulation develop. Naturally such disturbances eventually extend to the interlobular tissue, and the larger vessels circulating through them, but far more slowly; this is followed by neoplastic processes in those tissues and the eventual sclerosis of the interlobular tissue, which gives to these lungs, or portions of the same, the close resemblance to the lesions seen in contagious pleuro-pneumonia, and caused the mistakes which have been made in diagnosis by the British inspectors.

The "Humboldt lung" illustrates still another phase in the development of these processes in the lungs of corn-stalk disease cattle. A slower developing, more chronic course, in which the disturbance of the circulation occurred so slowly that necrobiotic processes seem to have been avoided and no real necrosis took place; here neoplastic conditions almost universally prevail and the complication of the interlobular tissue is much more evenly distributed, more densely sclerotic; in fact, much more resembling the conditions common to contagious pleuro-pneumonia, but with this difference, the disturbances all begin within and extend from the blood-vessels in the corn-stalk disease, while in contagious pleuro-pneumonia they begin and extend from the lymphatic circulation, especially the great lymph-sinuses in the interlobular tissue. This again accounts for the irregular complication of the interlobular tissue and the formation of lymph-cysts or pockets in the same in many cases of the corn-stalk disease, as illustrated by the Lord case, and I think, also, in the case quoted from the report of the Bureau of Animal Industry. The obstruction to the circulation of the blood and its sequential coagulation does not take place evenly in the blood-vessels; in fact, they are not distended evenly, but in spots here and there; perhaps at points where a branch is given off, or vessels suddenly turn in their course; perhaps at spots where the germs have collected in mass; at any rate the inflammatory centers along the blood-vessels are at first scattered and extend irregular from them; the sclerotic developments in the interlobular tissue follow the same course, thus irregularly (at first) encroaching on the lymph-sinuses, causing their complete obstruction in places and the formation of these lymph-pockets, which in the fresh specimens frequently resemble cysts filled with a more or less clouded fluid; section of the fresh lung frequently passes so close to them as to expose the thin wall filled with fluid containing pure cultures of the specific germs; when the animal has been dead a long time this fluid is frequently coagulated; in alcoholic specimens it always is, and forms a solid white mass which can be easily removed *in toto*, exposing a smooth-walled cavity to view.

In the corn-stalk disease, my experiences are unfortunately limited, I have found the form of broncho-pneumonia mentioned, and not the specific bronchial infective type seen in swine plague. Considering that the manner of infection is of necessity by way of the intestinal canal, it is very doubtful if the bronchial-infective variety, due exclusively to the germ of this disease, ever occurs. But another form of broncho-pneumonia does occur in this disease, due to adventitious germs, among which is the government swine plague germ, capable of developing in the catarrhalic-infected bronchioles in the manner described, but which had no more connection with the disease in question than if the animal had been afflicted with verminous bronchitis, and exposed to dust of some kind in which such a germ was suspended. These occurrences are more common in the septicæmic diseases of our domestic animals than the observers of this country and of Europe seem to be aware of. In sections of such lungs, sometimes one variety and sometimes several varieties of adventitious germs may be found mixed up with the genuine organism, which latter may have had but little to do directly with the lesions in question, though it prepared the way for them. Ultra-bacteriological investigators go on a wild goose chase after such adventitious germs, while men of pathological common sense cast away such chaff and pay their attention to the disease as it is, valuing complications as they should be appreciated. In this disease, as in swine plague, we frequently find an entire absence of consolidation in the lungs. In the same way with the spleen; sometimes it is swollen, often excessively, at others not. But never failing lesions will be those of a hæmatogenic character, varying from capillary to coarse vascular engorgements and hæmorrhages of various dimensions. The diffuse capillary engorgement of the kidney, which may be considered as pathognomonic of the southern cattle plague, never occurs in this disease, and hence may eventually prove of differentio-diagnostic value should it be finally shown that a germ of the same class as that of corn-fodder disease also invades grasses and causes disease in animals grazing during the summer months. Acute lesions that will never be missed, aside from the disturbances of the circulation, are those of the dense parenchymatous glandular organs, which are essentially specific to the extra-organismal septicæmiæ, and vary in degree from clouded swelling to the most extreme grades of fatty degeneration, as has been shown in the necroscopical notes.

To sum up, then, the corn-fodder disease is an acute extra-organismal septicæmia, due to a micro-organism belonging to the class of ovoid-belted germs, to which variety of diseases also belong the swine plague, southern cattle plague, Wild-seuche, hen cholera, and yellow fever in man; but in no case are these micro-etiological organisms one and the same, but each is a specific entity capable only of causing its specific disease, under natural conditions, in those animals specifically predisposed to its action, from some utterly unknown but equally specific physiological idiosyncrasy peculiar to its species.

SYMPTOMATOLOGY OF THE CORN-FODDER DISEASE.

In endeavoring to portray the symptoms of this disease we come face to face with a question of exceeding difficulty, because of their very close resemblance to other diseases which occur in cattle in our western states, and even anthrax itself offers intra-vital phenomena which more or less closely resemble those presented by this disease, especially in the most acute form. This fact has led many veterinarians into most serious errors in diagnosis. The occurrence of this disease in the months from November to March must be of essential differential diagnostic value.

In the first place, as to duration. Like the swine plague, this disease may be fatal in twenty-four hours, or it may extend to eight or ten days, or some weeks before such a result occurs. It is not a universally fatal disease, as we now know better than ever before.

Primarily we have to do with an acute blood-poisoning disease, which, like all such diseases, is accompanied by a more or less excessive exacerbation of the bodily temperature, varying, so far as known, from 39° C. to 42° C., that is, from 102.2° to 107.6° F. Such an excessive rise in the temperature must necessarily be followed by equally severe changes in the parenchymatous organs of the body, and consequent disturbances of the circulation, which frequently lead to excessive circulation—changes in the lungs, often followed by pneumonia, and most insufficient oxidation of the blood; hence, under such circumstances one would naturally expect a much accelerated and often very weak pulse and increased respiration, which, when consolidation of the lungs is present, is also very labored; and eventually, especially in chronic cases, great emaciation. These disturbances of the circulation frequently extend to the brain, where engorgement and cerebral pressure occur, which, in some animals, takes on the form of "craziness," as the owners call it. The animals then bellow fearfully, and chase other animals, especially dogs, hogs, or fowls, but seldom human beings. This has led to the mistake of their being called "mad" at times, and to this disease being mixed up with so-called "hydrophobia" in cattle by some veterinarians. Other animals stand by themselves, or are depressed and loth to move. Separation from their companions is one of the first indications of illness. As nearly as I can discern, they can all swallow and all drink. This is a very important point to be remembered. As in swine plague and southern cattle plague, constipation is a very frequent occurrence, while laxity of the bowels also often occurs, and may be looked upon as rather a favorable complication. Red urine does not occur. The visible mucosæ are injected, and often have a yellowish-red tinge. It will be remembered that my inoculated steer drank all the time, and even ate a little, during its most ill days. That such sick animals should be disinclined to eat, and, often, to drink, is no wonder; but if clear water is placed before them, no difficulty in swallowing will be discovered. Milch cows soon slacken in their yield of the lacteal fluid, and frequently the secretion ceases altogether for a time.

DIAGNOSIS.—It has been already suggested that this disease cannot be mistaken for the southern cattle plague: first, because of the season of the year in which the latter occurs, and the absence of Texas cattle; and second, the longer period of approaching illness in the latter, and the general average of its duration, as well as the non-existence of hæmaturia (red urine) in the corn disease. Again, the outbreak in the corn disease extends very slowly over a herd, and continues longer under ordinary circumstances; or in other words, the certainty of exposure to infection of the majority of the animals in the same herd, at or about one and the same time, is much greater in the southern cattle plague than in the corn-fodder disease.

That it bears no relation to the "Wild-seuche" of Germany is shown by the season in which it occurs, the locality and circumstances under which it occurs, and the absence of enormous œdema. In fact, when cattle, horses and other herbivorous animals become unaccountably ill immediately after having been turned into a stripped corn-stalk field, and that illness is accompanied by the phenomena previously detailed, it may be taken for granted that it is this corn-fodder disease, and no other.

PREVENTION.

Prevention of the Disease in Corn and Other Fodder.—The illustrations of the disease in corn and descriptions of the same have put us in possession of that practical knowledge by which we should be able to invariably tell when the growing corn in our fields is infested with this germ so dangerous to our live stock. The question of prevention should be the simple matter of avoiding such corn-fields as places for turning in stock to do the post-harvest gleaning. From conversation with several farmers, I am quite convinced that even now some of them have quite distinct ideas of the manner in which the corn itself is affected, and that as we have obtained an exact description of this part of the story from the botanical side, we should now be enabled to totally prevent this disease in our live stock, so far as the gleaning of our corn-fields is concerned. But this still leaves us to face with several uncertainties which can only be settled by careful observation by farmers, and exact investigation by competent investigators.

First—Knowing that the corn-fodder is diseased, both as to the husks and leaves, the question is, Does the germ also penetrate the cob and growing kernel, and can they also cause the disease?

My own opinion would be to doubt it, had I not had a very singular experience with a single cob of half-grown corn. At the time I did not have suspicion enough to investigate, and was also very busy in other directions, and equally annoyed by the owner of a horse which ate some of this corn, so it was said, and it was assumed that the corn had been poisoned by a vicious neighbor. All that was brought me was a "nubbin" ear of half-ripe corn. This was fed to a rabbit, and the animal died in three days afterward. There was every indication of a blood-poison disease. Anthrax was looked for, but not present. The organs were taken by the owner to a chemist, but nothing found therein. Cultures were not made, and only a very casual examination of the blood for bacillus anthracis, as I was busy beyond endurance at the time, and have never had any assistant to whom I could turn over the many such cases which have come to my notice.

This case is simply quoted as a warning worthy of the attention of other investigators.

Second—Are grasses also infested by this germ? And if so, which? This last is a very important question; for if they cannot be, then such fields can be used for raising grasses. This can be best determined by actual experiment by intelligent farmers, who should turn such fields into grasses, including patches of millet and clover, and then feed a few cattle with each kind, without the admixture of any other herbaceous food.

It would be well if the experiment stations would make suitable arrangements in this direction with intelligent farmers, and bear the expense of using such an infected corn-field for experimentation in about the following manner:

1. It should be thoroughly cleansed of all refuse of the old corn crop, but not exposed to the action of fire.

2. A portion should be planted in corn, and if any stalks appeared diseased during the growing period, they should be fed experimentally, and under every precaution against accident from other causes, to cattle and also rabbits; the latter might be tried first, as enough is now known of the germ of this disease to enable any competent person to recognize it correctly. When the corn is ripe the ears should be gathered carefully, and all full ears separated form those incomplete in development. Feeding experiments should be made with both. After the crop of corn had been gathered, a few cattle should be turned into it, in order to avoid any error. Naturally each section of the field should be separated from the other, and if, as Prof. Burrill's letter suggested, such a field as one of "forty acres" can be pretty generally infested, such an one should be used for this kind of experimentation.

3. Various grasses, including millet, clover, and in fact all kinds used for feeding stock, should be planted in lots in such a field; one lot of each kind should be used for pasturage in the summer for a few cattle, while the crop of another lot should be preserved for winter use, and then fed to a certain number of cattle.

It is too much to ask any farmer to do all this at his own expense; but the interests at stake make it the imperative duty of the agricultural experiment stations to do it, this being one of the purposes for which they were created, though Nebraska is the single state that has done its duty thus far in this regard. There are an abundance of public spirited and intelligent farmers who will gladly support the work. In fact Mr. McKelvey has written me that he intends to follow out my suggestions as to grasses and millet with the field he spoke of in his very detailed letter, stating his experiences with the disease. Later advices from Mr. McKelvey go to show that there is no danger in such grasses raised on such land.

Wherever such a disease has occurred, every stalk and leaf on such a field should be destroyed by fire, and, until we know to the contrary, the field should be seeded down to hay. There is no question but what the infested remnants of the corn-fodder can, upon their decay, cause the further infection of the field by the germ thus becoming free and again developing in the soil.

The absolute necessity of tile or ditch draining the lower portions of corn-fields has been so emphatically indicated by every practical observation, that it would seem unnecessary to call further attention to it as a most essential factor in rendering our corn crop almost immune from the dangers of this disease and thus save the cattle also.

Prevention of the Disease in Live Stock.—In discussing this question I will limit myself to cattle, because they are the only species of our domestic animals in which the disease causes serious loss.

Some might think it strange if I did not say a word about treatment, especially the farmer, while my scientific colleagues might think it equally strange should I do such a thing, for medicinal treatment in any curative sense is the height of absurdity in any disease of this class. Yet a close study of the clinical symptoms of this peculiar disease, and some knowledge of its pathology, does show that the offering of purgative doses of a saline character (Glauber's salts) to cattle in the very early stage of the disease, as well as to every member of a herd in which some have become ill upon exposure to this disease in a stalk field, is a matter of prophylactic importance. No harm can certainly be done by the thorough cleaning out of the intestinal tract in the animals still undiseased; and for those diseased, especially, and perhaps, also, those in the early stages, such a method of treatment is most certainly indicated as a possible means of equalizing the disturbances in the vascular system, and the avoidance of cerebral and pulmonary complications, as well as a tendency to deplete the blood of some of its septic elements and check the supply by the removal of so many of the specific germs as must necessarily occur in such a cleaning out of the digestive tract.

Such treatment, however, must take place within quarantined limits only, so that all the manure, and litter soiled thereby can be destroyed by fire when the outbreak is over. It must be borne in mind that if the manure and litter from a cattle yard where animals have had this disease is taken out and strewn over a field and then plowed in, and that field is planted with corn, that such corn is very liable to become invaded by this germ, and thus can be the cause of more losses in cattle if turned into such "a stalk field" the ensuing fall and winter.

On the eruption of this disease in a herd of cattle which have been used to do the lazy man's gleaning in a stalk field, the first step to be taken is the peremptory withdrawal of the herd from such a field and such fodder. The next thing to do is either to number, brand, or adopt some other means by which a record can be kept of each animal in the lot, and then take the temperature of each one night and morning. All with a temperature of over 102° F. must be looked upon as suspicious. Those in which it does not exceed or rise over 102° F. need not cause any worry. There is no need of separating the sick from the well, as the disease is not contagious. As mentioned previously, a saline purgative is indicated for all the animals. Those that die should be cremated, and with them a lot of the litter in the yard. If possible to avoid it the regular cattle yard should never be used for such cattle after any of them have become ill. Again, I repeat, in no case should a particle of the manure or refuse from a place where such cattle have been confined ever be used for fertilizing purposes. Burn it up, as well as the animals which die.

A SUMMER SEPTICÆMIA IN STOCK.

Ever since I have been in Nebraska, there has been a peculiar disease, and perhaps more than one, principally in cattle, although affecting horses, occurring in the hot months, particularly the latter part of June, July, and August. Whether these outbreaks are all one disease, whether or not some of them may not be anthrax, I cannot at present tell, never having seen a case myself. The outbreaks seem to have one thing in common in their etiology, however. They all occur on bottom lands, or lands in which there are large stagnant pools, or that have been seriously flooded in the late spring. The clinical phenomena so strongly resemble those seen in acute cases of the corn-stalk disease that some of the most prominent and competent veterinarians in the country have written me that they have seen cases of that disease in summer, and one, Dr. Knowles, of Terra Haute, Ind., wrote me in 1890 that he had seen a germ in "blood smears very much like that of corn-stalk disease." The time of occurrence of the latter disease, late in October to March, however, absolutely excludes it from being mistaken for this disease, as well as the fact of its constant connection with turning stock into stalk-fields. In the summer of 1891 a severe outbreak of disease in cattle occurred about Emmetsburg, Ia., and was visited by the Agricultural Department's agent, Dr. F. E. Parsons, who is not a bacteriologist, and so far as I know, nothing came from his investigations. I am also informed that the veterinarians of the agricultural college at Ames, Ia., also endeavored to investigate the same disease with similar results. Having made no personal observations of the disease in the field, and only having very incomplete information, as in all such investigations, our knowledge of this disease will have to be built up by fragmentary additions. For want of a more explicit nomenclature I have, then, termed the disease a "summer septicæmia," to distinguish it from the "corn-stalk disease," which has another origin, and is undoubtedly a winter septicæmia, which sharply differentiates it from this disease, as previously stated, as well as does the germ found in this disease, thus far, which also differentiates it from anthrax. Its symptomatology equally differentiates it from the "mad itch," in which the animals lacerate themselves frightfully by rubbing against wire fences or any object they can rub up against. This disease, again, acquires greater interest, from the fact that in recovered cases, which might be imported to Europe, the exposures of the ocean traffic might also give rise to secondary complications in the lungs of cattle, that may or might be mistaken for those of contagious pleuro-pneumonia, as has been done so frequently by British veterinary inspectors in the past, but in part, at least, have been demonstrated to be secondary complications following on the cornstalk disease by Nocard in France, and Bowhill in England.

A disease very similar to this occurs in the river bottoms of Nebraska and is termed the "bottom disease" by the farmers, but I have as yet been unable to receive material from exactly such localities. The material sent to me, however, did come from animals which were grazed on lands that were heavily flooded during the profuse rains of this spring, and occurred after the extremely hot weather set in in nearly every case. There were also stagnant pools in the pasture. The symptoms presented by the animals are briefly stated by our correspondents, who were the direct losers of stock, to be as follows:

Duration, two to four days. The animals are at first restless and roaming about the pasture from place to place, lowing and bellowing; they lose their appetite very rapidly and show signs of emaciation, continue in this restless condition and grow weaker, finally becoming delirious and chase any new objects, like strange horses or cattle, and are dangerous to strangers. They seem to develop a morbid appetite at times, gnaw the fence posts, and one steer was seen to eat of a dead hog which was in a putrid condition, and all seem to desire to gnaw something. They have considerable fever and in some a peculiar twitching of the skin has been observed. Some have diarrhœa and strain hard, passing small quantities of fæces at a time, which are of a very dark color and offensive odor. Towards the last they become constipated. As in such diseases, the contents of the manifold are very hard and dry in many cases; intestines congested and the other organs pale. In horses the symptoms are about the same.

The above is a summary of the reports given by farmers who have suffered losses, all of which agree in every essential particular.

From several outbreaks I have received blood taken by farmers, under as careful precautions as they could follow, from the jugular veins of killed animals (not dead ones), by first scrubbing the skin most carefully with soap suds and hot water and then washing it with fresh hot water. The knife used to open the vein had been first placed in scalding hot water. The bottles used were sterilized ones sent in mailing boxes from the laboratory and especially made for this purpose. The success of these farmers in following the directions and obtaining blood which grew absolutely pure cultures, even by plate tests, has been very encouraging and shows their interest in trying to aid the investigations of this laboratory as intelligently as they possibly could.

This germ is about the length of the swine bacillus, but much thicker, with rounded ends. It generally colors throughout of one shade, but during process of development uncolored spaces are visible in the body of the germs, and seem but separations of two organisms. It varies much in size, from an ovoid, almost round, object to one three times the length usually seen in the blood of the animal, many being curved somewhat like a new moon when these long forms are present. It is very mobile, resembling the swine plague germ in its movements. It does not fluidify gelatine. Grows well on agar-agar, on which it assumes the large forms when that medium is fresh. It grows well on coagulated egg albumen, the colony being almost imperceptible as it is of the same color. Here the germs are quite small and less in transverse diameter than on other media. It also grows on potatoes, in a gray-yellowish-white colony, scarcely perceptible in color at first, but growing darker with age. Also develops freely in bouillon, with no tendency to surface growth when freshly inoculated. Apparently the same germ is in small pieces of the liver, spleen, and kidneys of an animal killed in the last stages of the disease, the pieces being put at once into alcohol when cut out. Rabbits and guinea pigs are apparently far less susceptible to its action than the other germs we have worked with here. Pigs are not susceptible, if one can judge from one trial with one cubic centimeter.

Inoculated a twelve-months-old, very hardy and very fat steer with six cubic centimeters of a bouillon culture. The animal was seriously affected, but did not die. The following temperatures will show the effects:

July 5, 4:00 P. M.	102.50
July 6, 5:35 A. M.	105.00
July 6, 6:25 P. M.	106.60
July 7, 6:00 A. M.	105.40
July 7, 7:00 P. M.	107.60
July 8, 6:15 A. M.	106.80
July 8, 7:50 P. M.	107.00
July 9, 6:00 A. M.	104.90
July 9, 7:30 P. M.	105.00
July 10, 6:00 A. M.	102.50
July 10, 6:20 P. M.	104.40
July 11, 6:15 A. M.	103.40

On the 9th blood was drawn, under all necessary precautions, from the opposite jugular vein to the one into which the culture was injected, and the same germ found in the blood.

The practical prevention of this and similar diseases all lies in one act which every farmer can and should do. Like anthrax, this disease is dependent on a certain overmoisture of the soil, and develops primarily only in such localities. Practical results have demonstrated that certain localities that have been known for years for their murderous danger to stock as centers of anthrax infection have been rendered perfectly safe for grazing by complete drainage of the soil. It is the same with this disease, and the "mad itch." Farmers must drain first, and then fill up stagnant pools and sloughs where water collects, and also places in their grazing grounds which are unduly moist, if they would keep their stock free from diseases of this class. The one general rule is, and must be followed, drain and fill up all such places. There is no excuse for these losses. For years all intelligent authorities have been insisting on this one general and self-applicable rule of prevention for this class of diseases, but the majority of farmers seem to have entirely ignored this instruction. In plain truth, they deserve their losses, for they are due to their own neglect of every principle of common sense. These stagnant, death-dealing pools and moist spots are the graveyards of our stock on too many farms in the west, and must be removed. The remedy is simple and easy. No one but lazy men or an ignoramus will allow such places on his farm. Drain dry, and clean up, are the two essential preventive measures which every farmer can himself apply.

A SUMMER SEPTICÆMIA IN CATTLE IN IOWA, PROBABLY IDENTICAL WITH THAT PREVIOUSLY DESCRIBED.

(Contributed by Dr. T. D. Collins, D. V. S., of Emmetsburg, Iowa.)

On the bottom land on the west side of Des Moines river, about five miles southwest of Emmetsburg, which is on the east side of the river, several small herds of cattle owned by farmers in the vicinity were pastured during the summer of 1891. There were no division fences or other lines of demarkation dividing the feeding grounds of the different herds, except a furrow plowed to mark the lines or limits of each range. All the cattle of the different herds had, or could have had, access to the Des Moines river for water (always running water). All this bottom land—even the lowest draws—through which water first overflows during freshets, and also through which the adjacent country is drained into the river, and on which coarse heavy grass grows, similar to that found growing around sloughs on the upland, which are quite common in this part of Iowa—as well as the higher parts on which a shorter and finer grass grows, has a porous sand and gravel subsoil many feet in depth and coming to within four to twelve inches of the service. When the river is within its banks and the water in the channel below the level of the adjacent land, which is always the case except short periods during spring freshets, or following very heavy early summer rains (sometimes several years pass without an overflow from the river, but of course every year there is more or less wash through this bottom from the adjacent farms on uplands), the water on the bottoms rapidly soaks down or falls through this gravel to a level with that in the river. Natural conditions drain this land as effectively as it can be done.

During the seasons of 1890–91 the river banks were not overflowed, and there was no wash over the bottoms except what came from the table-land, ten to thirty feet higher than the bottoms, further back from the river, and all either in cultivation or meadow land, and water flowing from the farms on the table-land reached to the bottoms through depressions, or little ravines, in some places making a channel or washing out holes, and then either following the coarse grass depressions in the bottom before alluded to, or spreading out without a well defined course on its way to the river. So much for mother earth and natural conditions. And right here I think it well to draw your attention to conditions in the past, which I know will be recognized as a long familiar acquaintance, still always interesting. On the farms in the vicinity which drained over this bottom, to the river, hog cholera had prevailed to a greater or less extent during the previous twelve or fifteen years. Not always with equal virulence, still never entirely disappearing, and often very general and malignant and causing very severe losses, as this is a hog, cattle, and corn country. Our state authorities never to my knowledge having suggested or provided any prophylaxis to meet the case. On some farms the fowls would also rapidly die off, and up to about ten years ago there were frequent outbreaks of black-leg among the cattle, and in nearly all cases the carcasses of the hogs and cattle were not buried, but hauled out in some low place, or onto the bottom, and left on the surface to be devoured by other animals, dragged around in fragments by dogs, or left to decompose where they lay, and the next spring or summer freshet would wash over the ground, carrying the debris over the bottom land on its way to the river.

Now this particular outbreak of disease was confined to the cattle of one, and only one, of the herds feeding on the river bottom in that vicinity. This herd numbered about 175 head, and was composed of cattle of all kinds and ages. The first symptom of disease was observed on July 14, 1891, the weather being very warm at the time, and of this number over a hundred had died September 20. Showers and cooling of the weather seemed to abate the disease, and hot weather to intensify it and increase the number attacked. Some few died after October 1, 1891, and in two cases animals were affected and died late in November and December. After quite severe frosts, and fully three months after being removed from the bottom pasture and fed on the farm, I never heard of an instance of corn-stalk disease in this vicinity, or any other autumn malady among cattle. These cattle were owned by at least six different farmers, one of whom claims that some time after taking his cattle off the bottom and feeding them at home on his farm, where they had an abundance of good well water to drink, a young cow that had been in the herd developed the disease but did not die, making a partial recovery, remaining unthrifty, though given extra feed and care, and still so continues. This is the only instance in which it is claimed that an animal once showing the usual symptoms of the disease survived the attack, and as this owner likes to have a chance to express opinions differing with others, I think he is, to say the least, mistaken. From my own observation, and the statements of all the other owners of cattle in the ill-fated herd, I believe every case of attack proved fatal in from two to five days after first symptoms were noticed. I have never seen, nor do I know of, any other disease so surely and speedily fatal and involving in so short a time so great a percentage of a herd. No mild attacks and recoveries or chronic lingerings in this case. During this time not a single animal was sick or died of this or any other disease in any of the adjoining herds, although this diseased herd passed almost daily over a part of the feeding ground of one or more of the other herds on their way to Des Moines river to water, the land occupied by this herd being cut off from the river by the feeding ground of other cattle over which they were daily driven to the river for water. It may also be well to state here that on this ground, lying between the pasture of the diseased herd and the river, and on which other cattle ranged and fed all summer, there were two deep pools or washouts containing stagnant water which very seldom, if ever, dried out down to or below the level of the water in the river, and, owing to the deep gravel subsoil before alluded to, affected by the height of the water in the river. And furthermore, it is claimed by some, and by others denied, that prior to the outbreak the diseased herd, or rather the herd that became diseased, were confined to the pools for drinking water, and not allowed to go to the river that had then through recent rains further north toward its source become bank full, und the person in charge of the herd feared they might swim across the river and that it would be difficult to drive them back. Be this as it may, the cattle of the other herd on whose feeding ground these pools were situated had at the same and all previous times, and until about August 1st, free access to them, and every one acquainted with the habits of cattle knows how frequently they will reject the purest and freshest of water to drink from a pool or ditch by the roadside, from water that appears to man filthy and revolting, while these pools could hardly be called stagnant in its true sense. Still not one of these cattle sickened or died. The water in these pools may be loaded with impurities and reeking with filth, bearing the seeds of death, which was quite possible at least, and you will doubtless say probable, when I tell you they lay right in the track of the wash or drainage from the before mentioned carcass strewn farms and ravines. There may be myriads of deadly germs floating in the water or lying on the bottom, and from there carried out by the feet of the wading cattle and strewed over the surrounding ground and grass to pass from thence into the physical economy of temporary intruders, and caused the death in a short time of over 60 per cent of their number, while not one of what we may justly call the constant occupants of the premises was visibly affected, although they certainly fed around and drank from these pools until they were enclosed, about August 1st, after which time no cattle had access to the water in them, still the cattle occupying the ground on which they were situated, fed around them during the whole season with perfect safety.

I have dwelt on the existence of these pools and given you the incontrovertible proof regarding their relation to the different herds feeding near them. I do so, because as the culmination of a very dismal and unpopular farce in the way of an investigation by our state veterinarian, and an assistant, and aided by the doubtless equally learned and competent microscopist or bacteriologist who holds down the chair and draws the salary of assistant veterinarian to the experiment station, and who wisely agreed with his associate and their chief that the trouble was due primarily to the water drank from these pools, and secondarily, or rather finally, a perversity on part of the omasum, for which idea they may thank a fervid imagination, aided by the timely suggestions of some local quacks whose intelligence consisted of the inspiration drawn from bottled spirit, they conjointly advised the farmers that the immediate and direct cause of trouble was impaction of the omasum, and relief was to be had by cathartics, physic, physic, physic, and so it went, until all the procurable salts and oils in the vicinity from five or six drug stores was exhausted, and greater supplies ordered and used from day to day, with, of course, no visible effect, but an increase in virulence of the disease as the weather became hotter and the cattle depleted by purgatives. What a haven of refuge for ignorance in the diseases of ruminants is that peculiar and innocent organ the omasum!

A short time prior to this outbreak, there had been a mad dog scare in the county about fifteen miles distant, and a man who had been bitten by a dog there was then in Chicago receiving the Pasteur treatment for rabies, and on July 15, 1891, a man owning part of the cattle in this fated herd, and provided with the usual cattle chasing tramp dog, story, called my brother to examine the herd, saying he "feared some of his cattle had been bitten by a mad dog, and now had likewise gone mad." I accompanied my brother on this call, and on examination found that the description of the symptoms given us relative to delirium and violence toward other animals was very much overdrawn. We found one animal dead, and a few others affected, one of which died within an hour after we reached the herd.

I have carefully studied the article entitled "Another Germ Cornered," and published in the *Nebraska State Journal* of July 23, 1892, and was not a little surprised on finding how closely the description of symptoms, a knowledge of which had only been acquired by you by report, agreed in a general way with what we observed in the diseased animals of last summer. I will now review the description contained in that article in the *Journal* and make such comment as I find necessary from the standpoint of our observations and examinations among the dead and dying cattle of last summer. The opportunity was certainly all that could be desired by a specialist qualified to make a proper investigation and provided with the necessary apparatus. I heard some of the owners of the diseased cattle tell the veterinarians sent there by the governor of the state that if they would stay and endeavor to find out the cause of the disease they could have the privilege of destroying for examination any or every animal affected, at any time after first symptoms were observed. In this way they could have fresh subjects every day for several weeks, but the opportunity was thrown aside and lost. Then an effort was made to induce the governor to apply to Dr. Billings to, if possible, come and investigate the matter properly, to which he replied that he was willing and anxious to further and aid, to the limit of his authority, a thorough investigation of the trouble, but that he had no authority under the laws of the state to incur any expense in the employment for such service of any one living outside the state. So you see we have a snug little officio-scientific combine here, neatly fenced, so as to safely enclose indifference or ignorance. Then, on application to the United States Department of Agriculture, Dr. Parsons was sent here on a mission of inquiry and observation, and there the matter dropped, and so did the cattle daily in death. Now for the symptomatology: Your correspondents give duration of the disease at "two to four days." Here we might say two to five days, being most rapidly fatal in the hottest weather. We also found the first symptoms were, restlessness, a tendency to separate from or hang around the outskirts of the herd; would stop or start off suddenly; lie down for a considerable time, then get up suddenly and run or walk fast in a nearly direct course without any apparent object towards which they were going, then stop suddenly, and then, quite likely, return toward the herd. While I many times noticed them run towards other cattle, or dogs, or hogs that may come near as if to attack and horn them, I never saw them use any great violence in doing so; usually, they would stop on or before quite reaching other animals of their kind, shake or drop the head, breathe rapidly, stand still with an alarmed, or, I might say, apprehensive appearance, as if intently watching or listening to something. From the earliest stage appetite was suspended, neither would they drink when driven to the river, though frequently the nose and head up to the eyes was in the water, none was swallowed. Still we never found any cause that would obstruct or prevent deglutition, and they would always swallow liquids from a drenching bottle. A dismal sounding lowing or calling was kept up from an early stage until death. But little diarrhœa was observed, and that in the earliest stage, soon followed by constipation, gauntness, restlessness, weakness, and very marked and frequent tenesmus, which increased in severity and frequency until death, as may be expected under these conditions and a temperature running from 103° to 106°; the quantity of fæces excreted rapidly decreased in quantity, and became hard, dry, and stick-like in form, less than an inch in diameter, very dark or greenish-black in color, showing longitudinal corrugations, and covered with mucus and exfoliated epithelium. This coating increased in consistence and adherence as the disease advanced and the quantity of fæces decreased. Sometimes there was stamping, kicking, and looking back at the side as if suffering abdominal pains. The urine is scanty and somewhat high colored, with no peculiar odor, while the odor of the fæces is heightened and of a kind of musty, stale offensiveness.

Now as to this morbid appetite, said to be sometimes noticed, with a constant inclination to gnaw something on the part of all, as a feature in the symptoms described by your Nebraska correspondents, that was not at all observed in the outbreak here. It is true that very often, in its feverish restlessness, an animal would grasp a mouthful of feed, or any other prehensible substance near, mumble, or perhaps hold it a short time, and then drop it, apparently losing knowledge of its presence in the mouth; or sometimes nuzzle another animal if lying down, or nuzzle or rub its nose or head against posts, logs, boards, or anything else near, but this gnawing symptom as in rabies was entirely absent, as was also the reckless, delirious combativeness described to you, and also imagined by some few observers here. It is true that, in a very few instances, three or four milch cows ran around wildly in the last stage when the fever was the highest, and would chase or follow anything that would run from them, man included, still no other animal or person was injured by any of those diseased; while we know that in the fever and delirium of anthrax cattle are in many cases dangerously violent. In fact we did not observe either coma or delirium sufficient to warrant a suspicion of cerebritis or meningitis. The peculiar twitching of the skin, also trembling, we noticed. Likewise the abnormally dry and solid state of the food in the manifold, a condition which we know always follows loss of food and water even when in the best of health, awaiting slaughter in the stock yards. We could discover no pathological conditions in the tissues of the omasum, not even an odor. This was likewise true of the rumen and reticulum, and of the abomasum except in two or three cases in which we thought there was a slight congestion of the mucous coat; but sometimes, commencing at the pylorus and always in the duodenum and throughout the whole intestinal tract, but more especially in the small intestines, we found thickly distributed in the mucous coat patches (varying in size from several inches in length, down) of congestion, inflammation, or ulceration in different stages; also in several cases we found petechial spots on the serous coat of the intestines. In the mesentery, liver, and spleen (*never found any great enlargement of the spleen*), found some patches of ecchymosis in the mesentery near the intestines, also in a few cases in the liver, and in some cases found parts of the liver very friable, purplish-brown in color, and the capsules easily separated from the surface. And what attracted our attention most was the color of the blood. It was quite dark colored, even when drawn from the carotid artery. It would turn bright red on exposure to the air, and also clot quite readily, and there was *little if any congestion of the spleen*, still the blood was very evidently in a much altered condition, and without a proper supply of oxygen for the tissues of the body; another thing quite noticeable, on exposing the intestines immediately after death they had only very little vermicular motion, and least when the intestines were most affected, while in those slaughtered in health this involuntary muscular action is quite marked and continued.

This article is already perhaps too long drawn out, and doubtless in part irrelevant to the matter of investigation. Still as I know you would rather grope carefully through bushels of chaff than lose one kernel of fact, I must give you a few features and points relative to this diseased herd that are to me incomprehensible, and I think you will admit somewhat puzzling to account for.

Now bearing in mind the former outbreaks of hog cholera, blackleg, etc., and the disposition of the carcasses of animals dying therefrom, and their subsequent relation to the feeding ground and drinking water of the diseased herd, of all of which we had a personal knowledge, having lived in the vicinity and frequently passed over the ground every season during the previous twenty years, and having at the first visit to the herd, and on making an autopsy, observed the condition of the blood and intestines, and noting its rapid course and fatality, we diagnosed it a disease of a specific nature and closely akin to anthrax, and advised the owners not to depend on medical treatment, as either curative or prophylactic, and to at once change their cattle to other and higher ground. They claimed they could not, and certainly did not, follow our advice, with the result before stated. Now I will state a few cases that look like freaks of the disease. First, from whence came the cause? Remembering the proximity and immunity of *all* cattle outside this herd, even when subjected to nearly the same conditions, makes it difficult to locate it. Also a man named Stanzbach, who owned part of the herd, and lost over sixty per cent, had three work oxen which he turned out with his other cattle in the herd for about two weeks early in the spring, and after that time kept them on his farm in tame grass pasture and watered them from a well of pure water, intending them for fall beef. At night he yarded them with his cows brought from the herd, and in about eight to ten weeks after being on the bottom, two of the oxen died, showing the usual symptoms of the disease. This same man also lost a calf that had never been on the bottom, yarded not with, but adjoining the yard in which the cows and oxen were kept at night, only separated by a wire fence; did not suck its mother or any other cow for two months. It was fed with his other calves with skim milk from a creamery vat or tank in which the milk of this man's cows, and that of several others in this vicinity as well as the milk of many others outside of it, was put for return to the patrons of the creamery. No outside calves were injured by using the milk from this common tank. The calf had all the characteristic symptoms. Query—How was it communicated to this calf? Another calf owned by a man named Greeley, and allowed to run with its mother on the bottom, and which surely did not drink from stagnant pools, died, showing all the usual symptoms, and about a *week afterward* its mother sickened and died in like manner. Again, a cow taken from the herd late in September, and brought to Emmetsburg stall-fed and watered from a good well, died in December, about three months after being taken off the bottom, and after the occurrence of severe frosts and quite heavy snows, this animal showed all and exactly the same symptoms as those that died in midsummer. This leads us to think of the period of incubation and vitality in different temperatures. There were also two other cases almost exactly similar to the last described.

During Dr. Parsons' visit here we learned that a similar disease in a bunch of about seventeen hundred cattle kept about twenty miles north of here and known as the Petijohn herd. Dr. Parsons went up there and found the herd was that day removed. They were started southward toward Sac and Buena Vista counties, where the cattle were owned by many different farmers. Dr. Parsons had one of the losers of the cattle near here with him on his trip north, and they learned that at least sixty or seventy head had died within a short time previous, which alarmed the herder, and he started the cattle home to their owners. (These cattle were feeding on a large tract of unsettled common prairie, containing some sloughs or water holes. It was neither marshy nor hilly land, but rather level or gently undulating prairie, and of course free from the conditions resulting from hog cholera and anthrax that existed here. I don't know that this big herd had any running water or wells to drink from. I think they were dependent on natural prairie ponds and sloughs. The land was rich, black mould surface, and clay subsoil. Next day following Dr. Parsons' trip north, we intercepted this big Petijohn herd near Ruthven, twelve miles west of here, and there found animals exhibiting exactly similar symptoms to those of the sick cattle near Emmetsburg. We learned from the men in charge that the duration of the disease was the same. Every case proved fatal, and in fact agreed in every particular. We observed animals in different stages of the disease. One lay dead, and they had been falling by the roadside on their march home. I afterward learned that many of them died after being returned to their owners, at least sixty or seventy miles from where they were herded. I have also heard of some other instances of this or a similar disease, but only wish to describe those of which we have personal knowledge. We are carefully watching for the occurrence of a new case here that we may be enabled to send you some material to aid your investigations.

With this perhaps too detailed a description and history of this somewhat noted outbreak of disease, I will bring this long-drawn-out article to a close.

Before closing myself, I wish to express my sincere thanks to Mr. Grupe, the efficient draughtsman of the State Journal Company, for the interest and care which he has given to the illustrations.

ADDENDUM.

"THE BREEDERS' GAZETTE" ON THE AGRICULTURAL DEPARTMENT'S RELATION TO THE CATTLE INTERESTS.

The *Gazette* wishes just a few plain words with the United States secretary of agriculture upon a subject in which many of us have been deeply interested for a number of years, viz: The final extirpation of the last traces of the pleuro-pneumonia in cattle, to the end that an absolutely clean bill of health may be shown our foreign buyers. First let us recall a bit of history.

Some years ago the National Cattle-Growers' Association, representing all the organized cattle owners of the Union, formally demanded of the national congress the creation of a government cattle commission, two members of which should be experienced, practical cattle-growers of good executive ability and one a competent veterinarian, whose duty it should be to take immediate and vigorous steps toward the thorough eradication of contagious diseases. The general sentiment was one of distrust of the veterinary profession so far as prompt and thorough work was concerned. The measure in question was endorsed by every leading state and national organization of cattle-growers in the country. It was endorsed by the boards of agriculture of all the greatest agricultural states. It was endorsed by joint resolution in a number of western state legislatures and petitioned for by thousands of people who feared just the condition with which we are now face to face. It passed the senate and was favored by a clear majority in the house, as well as by President Cleveland, who stated in the presence of the writer hereof that he believed a commission thus made up could be relied upon to make shorter work of the plague than would be the case if it was left solely in the hands of the veterinary profession, and his experience with the matter while governor of the state of New York qualified him to speak intelligently upon this point. The measure, it is needless to say, was bitterly opposed by Dr. Salmon, then, as now, chief of the Bureau of Animal Industry, who, of course, regarded the bill as not only shearing the department of some authority, but implied as well some lack of confidence in the ability of the bureau to make short shrift of the disease. Chairman Hatch, who is also at all times jealous of the prerogatives of the department which he has labored so assiduously to build up, refused to report the bill to the house, and with the connivance of the speaker (now the junior senator from Kentucky) succeeded during the closing days of the session in defeating the will of a majority of the house and of congress. The responsibility for the defeat of this the most effective measure yet presented for the opening of foreign ports to American cattle was deliberately and openly assumed by the Department of Agriculture. The officials of that branch of the government positively declared that the bill was wholly unnecessary. "Only give us another appropriation," they urged, "and we will wipe out pleuro-pneumonia in twelve months." It is scarcely necessary to add that such talk did not deceive the cattle-growers. They believed that if their bill failed they would, in all probability, have the taint of disease left upon their herds for an indefinite period. Still, they lived in hopes, and when they saw heavy appropriations being made to enable the bureau to fulfill its promises they possessed their souls in patience. The months rolled by and the department's boasted ability to cope with the disease proved, as had been anticipated, a mere political bluff. Then came Gov. Rusk's accession to power, and the earlier days of his administration were full of promise of the long sought relief. He ordered sick and quarantined cattle killed in spite of alleged "constitutional" objections to such destruction of private property, and informed the owners that the sick cows had to "go," even if he had to call the military power to his assistance. This was like Jere Rusk. It looked like business. It was exactly the line of policy the proposed cattle commission was to have followed. The whole country applauded, and it looked as if the end of the pleuro was at last at hand. But lo! a change came o'er the spirit of the secretary's dreams. His good right arm fell nerveless to his side. He succumbed to the same subtle power which the chief of the Bureau of Animal Industry has through succeeding administrations exercised over his superior officer. Years have passed since Colman, Salmon, and Hatch undertook their "twelve months" campaign. The head of the department has been lulled to sleep. The sick cow still survives the millions of money wasted in her farcical pursuit by the veterinarians, and Britain's gates are yet securely closed.

Mr. Secretary, there is no longer such a thing as disguising the profound disgust with which the farmers of the west now view your department's management of pleuro-pneumonia. The dilatory policy of the Bureau of Animal Industry in this matter can no longer be patiently withstood by western cattle growers. A suspicion that your subordinates are purposely delaying the finishing stroke lest they should find themselves out of a job and unable to wrest additional appropriations is rapidly taking root, and they are looking to you to infuse a little of your old-time personal vigor into the work. Unless this suspicion can be thoroughly removed you may as well count upon another organized revolt from the cattle-growers against a continuation of bureau methods; and the record of facts since the Warner Miller bill was defeated by gag methods applied by the chairman of the house committee on agriculture is not such as will again avail in opposition to a demand to place this work in the hands of men who will handle the matter without gloves. We say this in all friendliness and good will. We simply assert that unless the last sick or quarantined cow is soon led out and shot that this matter will not only become something of an issue in politics before the ides of November, but will form the subject of some interesting debate when the next agricultural appropriation bill is reported in congress. We are sick and tired of waiting for the relief which your department pledged itself to speedily bring about if congress would but give the veterinarians "a chance." The day of grace has already been sinned away. Action is the only thing that will satisfy now; and it must be action prompt, vigorous, and decisive. We have been on the point of being declared free from lung plague so long that we now begin to fear that it is not the policy of this bureau to clean it up. Do you not remember that during the first week of your present official life you were warned that in order to effectually get rid of this disease it might be necessary for you to "sit down hard" upon the veterinarians who are living upon it? One decrepit cow serves to procure a new appropriation each year, but the old "snag" also serves to close all the ports of Great Britain to our healthy western herds. It is now for you to decide whether you will stand by the discouraged producers or with your dilly-dallying subordinates. When it comes to this choice of roads the *Gazette* cannot doubt that your energies will be thrown in the interest of the people.

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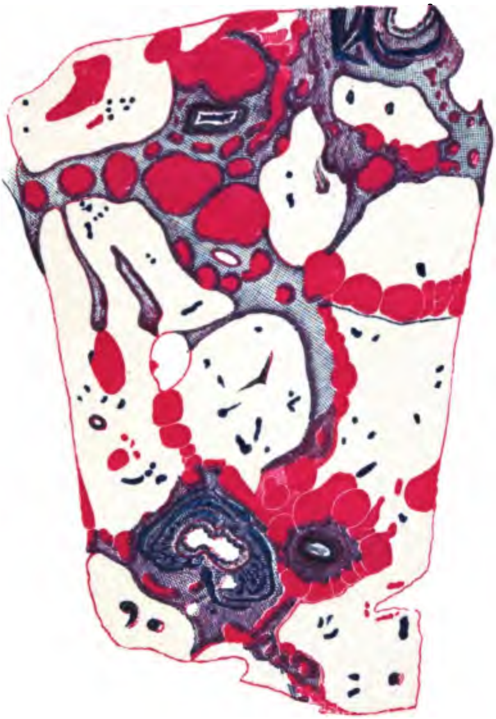

Coagulation obstruction of circulation causing necrotic pneumonia in Swine Plague.

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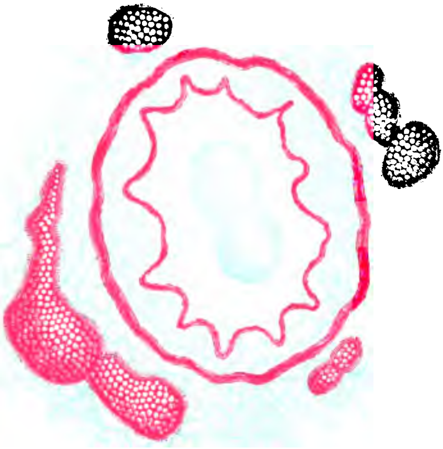

Coagulation, obstruction of the circulation in the lungs, illustrating the first stage of destructive pneumonia in Swine Plague.

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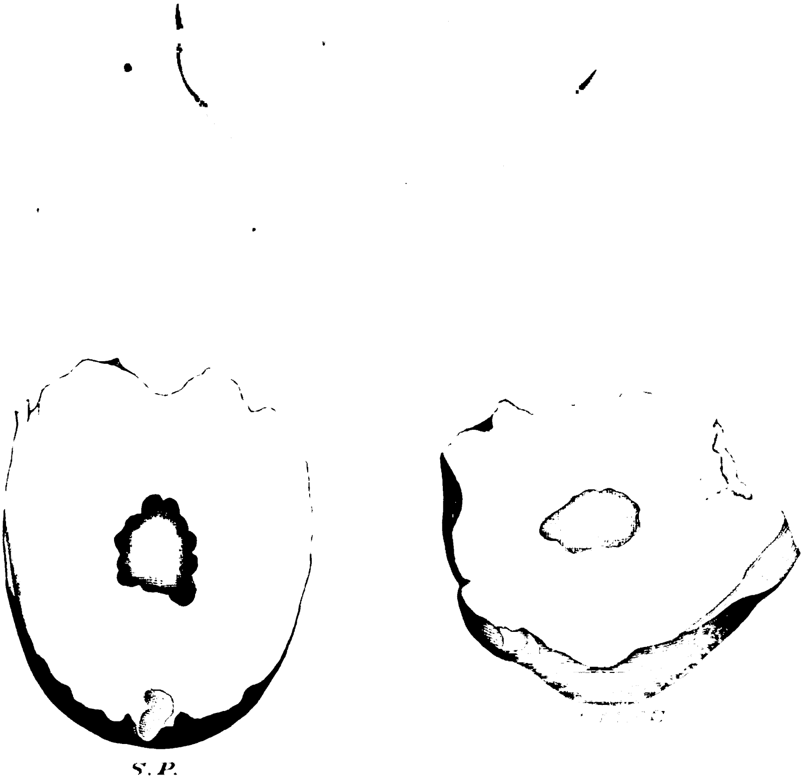

Growth of the Germs of Swine Plague, the Corn-Stalk Disease, and Southern Cattle Plague on Potatoes.

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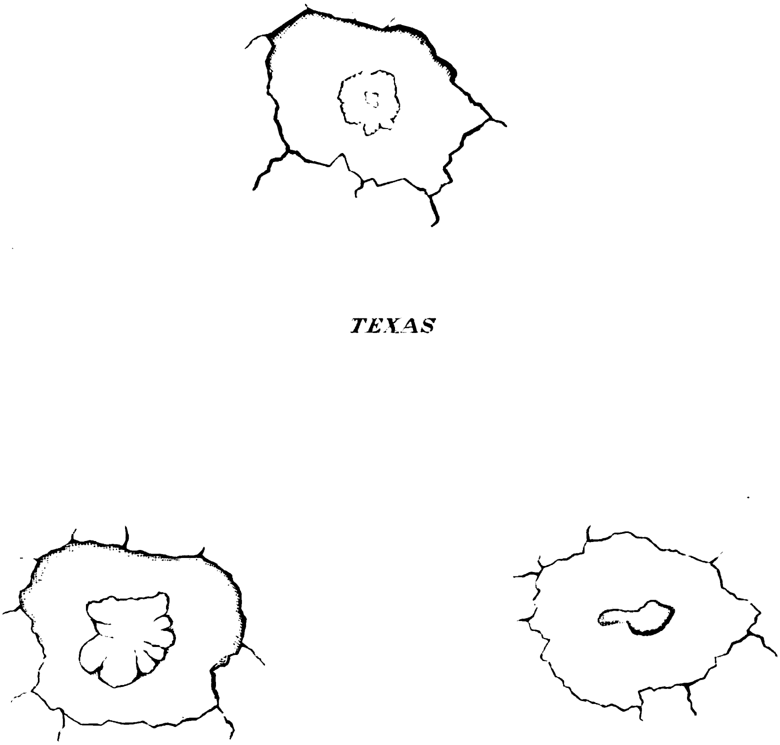

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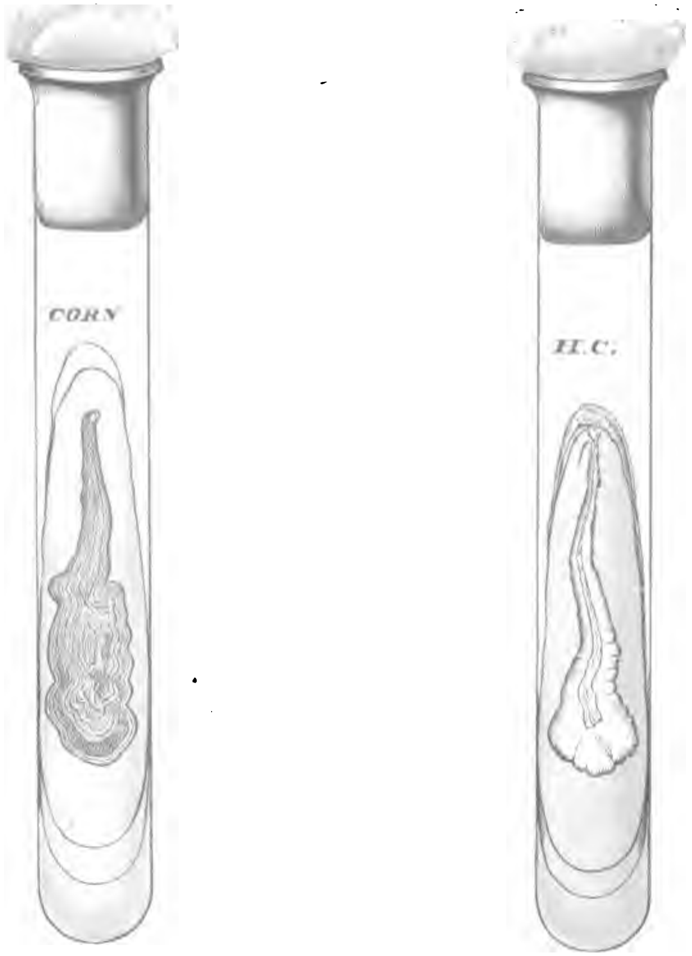

The Corn-Stalk Disease Germs and those of Swine Plague, as they develop upon Agar-agar.

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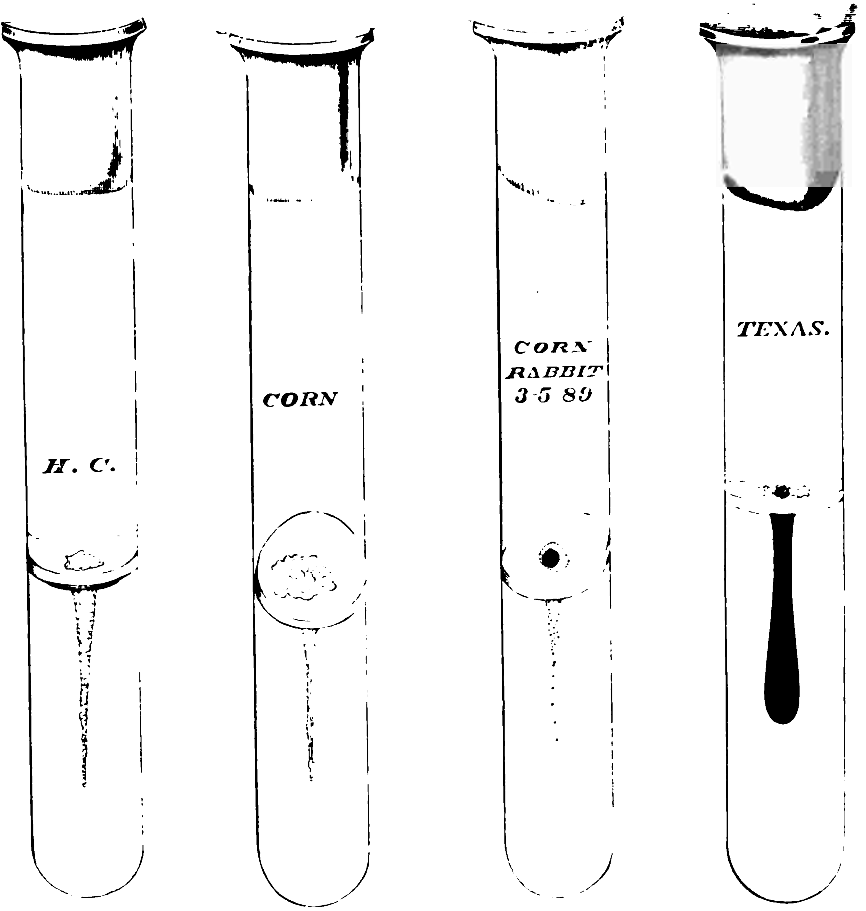

Puncture developments of the Germs of Swine Plague and the Corn-Stalk Disease in gelatine, the third tube being a fresh culture made from the blood of a rabbit having the Corn-Stalk Septicæmia; also, culture of Germ of Southern Cattle Plague fresh from body of tick in gelatine; only produces this pigment when fresh or carried in bouillon; loses this property in old cultures on solid media.

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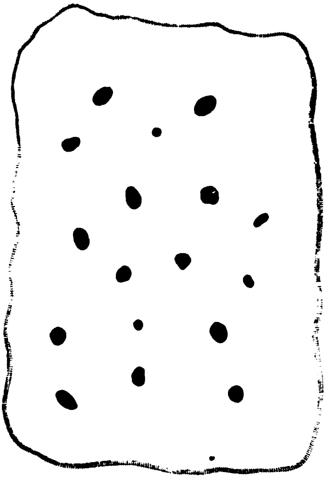

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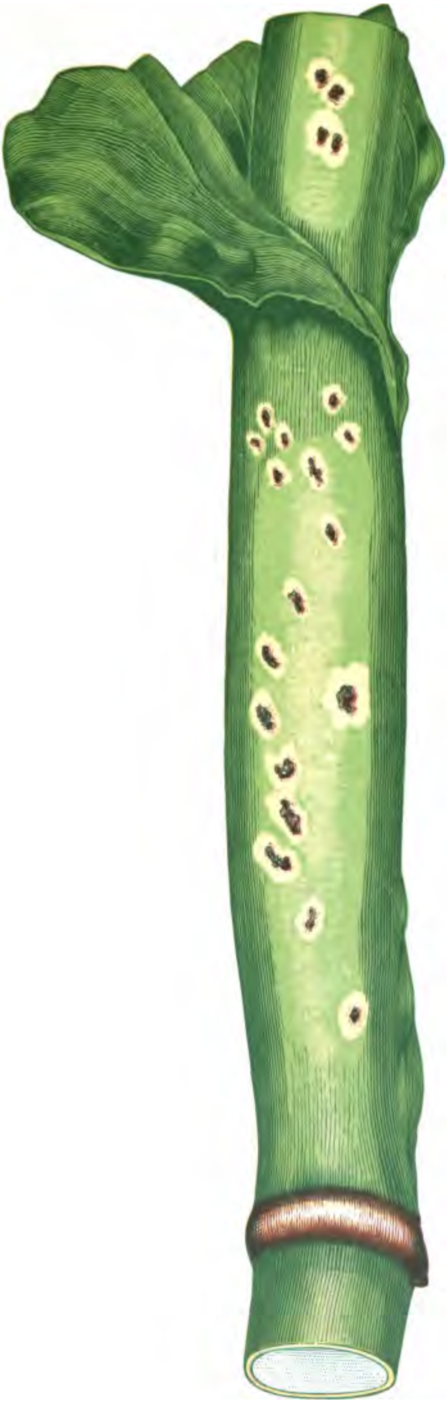

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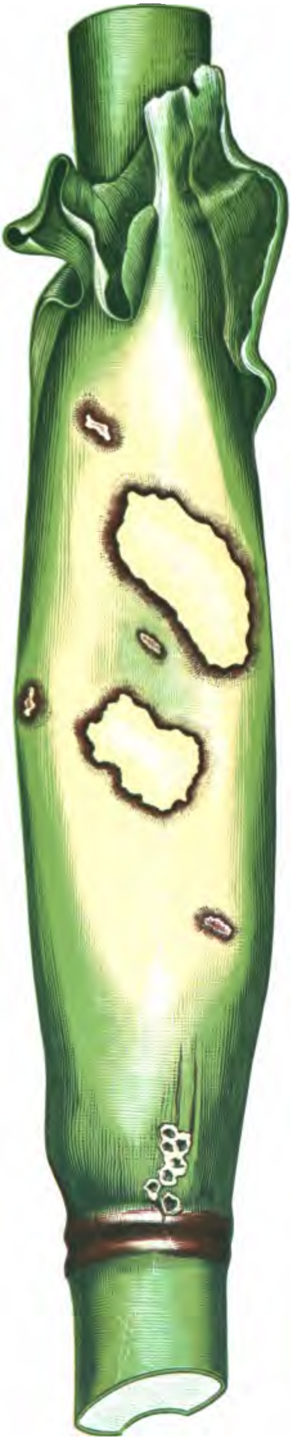

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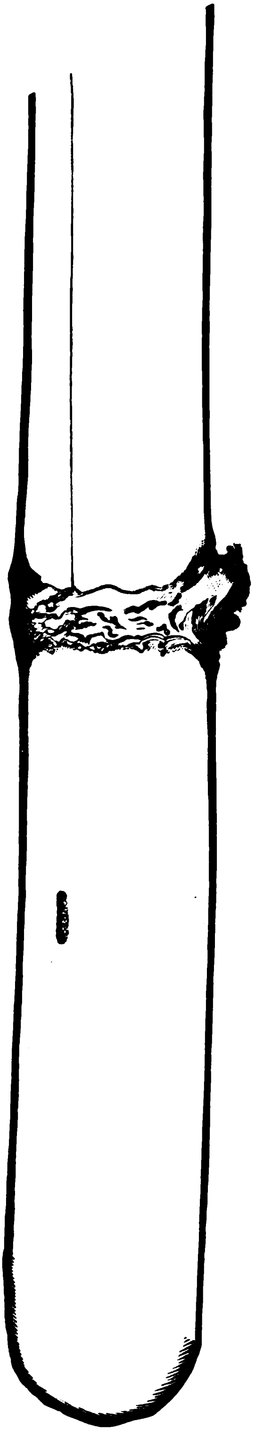

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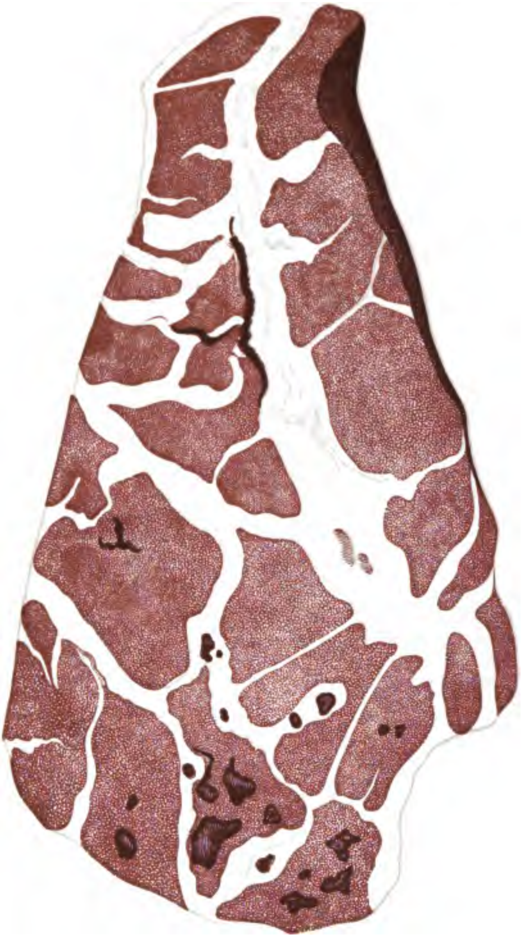

Sequelent chronic interstitial pneumonia in the corn-fodder disease, mistaken for contagious pleuro-pneumonia by British veterinary inspectors.

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